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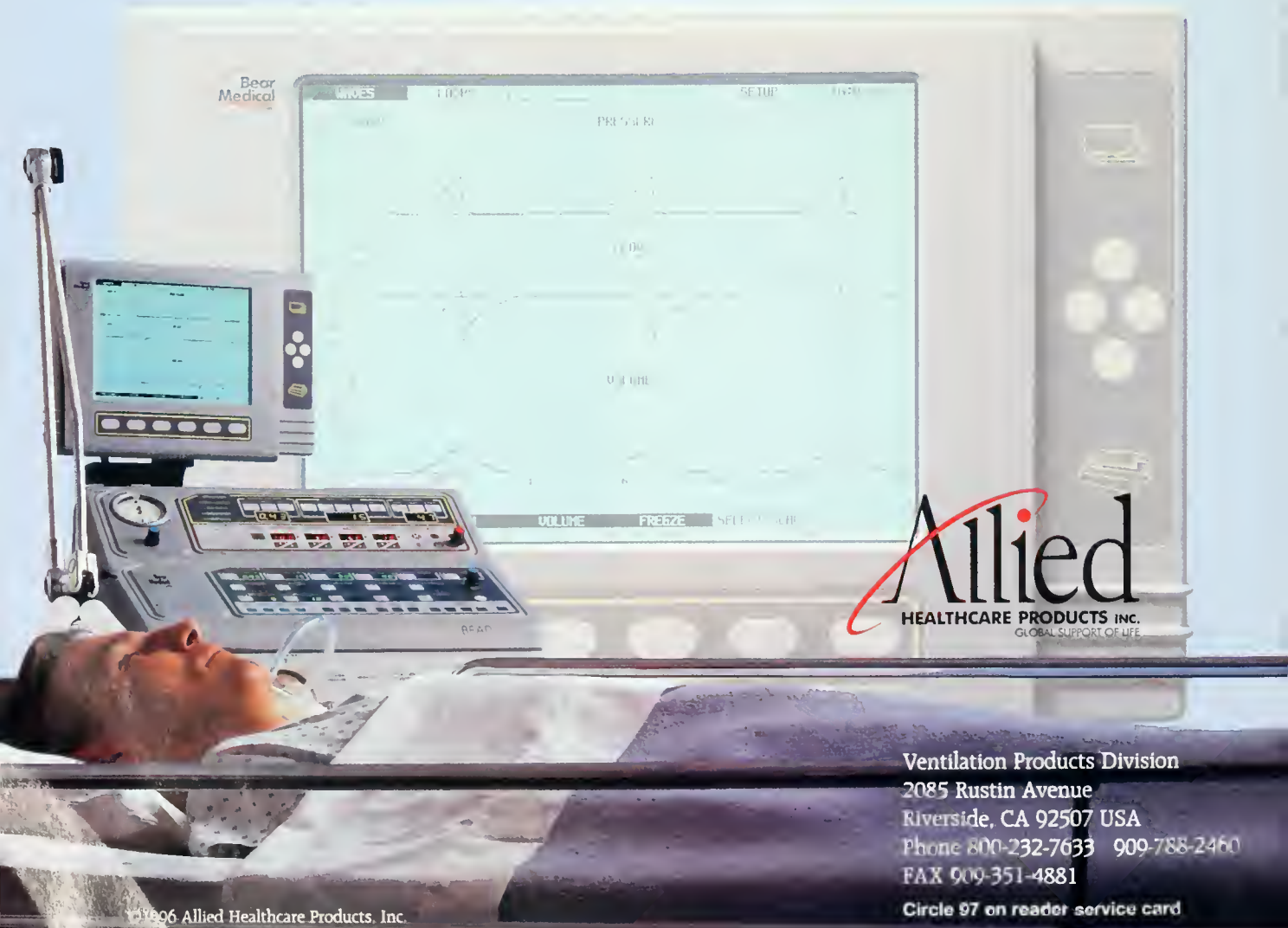

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CLASSIC REPRINTS

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by Julius H Comroe Jr—Reprinted, with permission, from the American Review of Respiratory Disease 1976;114(4):795-802

CORRECTION

49 Corrected publication information
Op't Holt TB. Comparative review of respiratory care equipment texts. Respir Care 1995;40(8):876-878.

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Editorials, Commentaries, and Reviews To Note

Pulmonary Rehabilitation in Patients with COPD (Commentary)—BR Celli. Am J Respir Crit Care Med 1995;152:861-864.

Beta-Adrenergic Receptors and Their Regulation (State of the Art)—PJ Barnes. Am J Respir Crit Care Med 1995;152:838-860.

Terminal Weaning (Debate in Print)—D Gianakos. Chest 1995;108(5):1405-1406.

Rapid Withdrawal of Support (Debate in Print)—T Gilligan and TA Raffin. Chest 1995;108(5): 1407-1408.

Quality of Life in Octogenarians after Open Heart Surgery—P Kumar, KJ Zehr, A Chang, DE Cameron, WA Baumgartner. Chest 1995; 108(4):919.

OBJECTIVES: To determine the quality of life in octogenarians after open heart surgery. BACKGROUND: Despite an increasing number of cardiac operations on octogenarians, the outcome as measured by functional status, independence of living, and psychological parameters of quality of life remain unproved. METHODS: Two groups of octogenarians (Group 1, undergoing operation in 1986; Group 2 in 1991) were reviewed retrospectively to determine operative mortality and functional results. RESULTS: Group 1 (n = 15, mean age of 83.2 years) and Group 2 (n = 53, mean age 83.0 years) were studied. Operations included isolated coronary artery bypass grafting (Group 1, 10, Group 2, 29) and valve replacements ± coronary artery bypass grafting ± other procedures (Group 1, 5; Group 2, 24). Group 1 had 9% hospital mortality and 53% actuarial survival after a mean follow-up of 6.3 years. Group 2 had 17% hospital mortality and 72% actuarial survival after a mean follow-up of 1.5 years. At follow-up, significant improvements were observed in New York Heart Association (NYHA) angina class, congestive cardiac failure class, number of cardiovascular symptoms, and indices for satisfaction with overall life and general affect in both groups. Further, Group 2 also showed significant improvements in independence of living, ease of life, and Karnofsky dependency category, but these improvements were less evident in Group 1 after a longer period of follow-up. At follow-up, 75% of Group 1 and 84% of Group 2 octogenarians would definitely have made the same decision to undergo open heart surgery in retrospect. CONCLUSION: This study demonstrates improved quality of life after open heart surgery in octogenarians.

Predicting Death after CPR: Experience at a Nonteaching Community Hospital with a Full-Time Critical Care Staff—L Bialecki, RS Woodward. Chest 1995;108(4):1009.

OBJECTIVE: To identify a series of variables that predict death after in-hospital cardiopulmonary resuscitation (CPR). DESIGN: Retrospective observational study. SETTING: A nonteaching community hospital with 24-hour on-site critical care specialists. PATIENTS: Consecutive adults undergoing CPR between August 1989 and July 1991. INTERVENTION: None. MEASUREMENTS & MAIN RESULT: Two-hundred forty-two patients suffered a total of 289 cardiopulmonary arrests. Forty patients (16.5%) survived to discharge. Thirty-nine (16%) patients had more than 1 cardiopulmonary arrest. Survival of second CPR was 18%. Acute physiology and chronic health evaluation (APACHE) II scores within 24 hours of admission and CPR [APACHE(a) and APACHE(b)] were measured. APACHE(a) and (b) scores more than 20 had a 96% predictive value positive and were associated with a 5-fold decrease in survival. Besides APACHE, cardiopulmonary arrests on medical floors and after day 4 of hospitalization, duration of CPR more than 15 minutes and asystole assumed significance at multivariate levels for predicting death. Ventilatory assistance and Glasgow coma score of less than 9 at 24 hours after CPR predicted death for initial survivors at multivariate levels. Survival on telemetry units were similar to the ICU (17 vs 21%) but twice that of the medical floors. CONCLUSION: The CPR outcome can be predicted early during hospital course, which may assist physicians to formulate a do-not-resuscitate order. Patients surviving a CPR should be considered candidates for another resuscitation if clinically warranted. Low-risk patients can safely be admitted to telemetry units instead of to more costly ICU's.

See the related editorial: ***Predictors of Cardiopulmonary Resuscitation Outcome in a Community Hospital*** *—SD Nelson. Chest 1995;108(4): 892-893.*

Work of Breathing as a Weaning Parameter in Mechanically Ventilated Patients—MM Levy, A Miyasaki, D Langston. Chest 1995; 108(4):1018.

STUDY OBJECTIVE: Predicting patient responses to changes in ventilatory support or weaning is primarily based on bedside measure of respiratory mechanics and clinical judgment. Work of breathing (WOB) has been considered to be the best predictive factor in weaning. This study evaluated patient WOB as a predictive measure of weaning and extubation. DESIGN: Blinded, descriptive evaluation of the accuracy of pre-extubation values of patient WOB in predicting successful extubation. SETTING: Critical care units of 500-bed major teaching hospital of the University of Hawaii. PATIENTS: Twenty-four patients from the medical and surgical ICU were enrolled in the study. Patient diagnoses included COPD, ARDS, pneumonia, and renal failure. MEASUREMENTS & MAIN RESULTS: In all patients, weaning from mechanical ventilation was accomplished by the clinical ICU teams who were blinded to the patient WOB. After extubation, patient WOB was compared with extubation attempts. In 14 patients, WOB was elevated above normal levels prior to successful extubation, while only 1 patient whose WOB was abnormal prior to extubation required reintubation within 24 hours. WOB and clinical parameters were normal in 10 successfully extubated patients. CONCLUSIONS: In this group of mechanically ventilated patients, measurement of patient WOB was less accurate than conventional weaning parameters and clinical judgment for predicting suc-

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cessful extubation. This clinical evaluation study suggests that WOB alone is inadequate as a weaning parameter. *See the related editorial:* ***Work of Breathing Measurements: Can They Help Identify Patients Who Can Be Successfully Extubated?*** *MJ Mador. Chest 1995;108(4):893-894.*

Elevated Imposed Work of Breathing Masquerading as Ventilator Weaning Intolerance—OC Kirton, CB DeHaven, JP Morgan, J Windsor, JM Civetta. Chest 1995;108(4):1021.

OBJECTIVE: To test the hypothesis that, if apparent ventilatory insufficiency observed during a weaning or preextubation trial is due to a significant contribution of imposed work of the endotracheal tube and breathing apparatus (WOB_{imp}), and the patient's actual physiologic work of breathing (WOB_{phys}) is not excessive, it should be possible to extubate these patients safely. DESIGN: Prospective descriptive study. SETTING: University hospital trauma intensive care unit. PATIENTS: A total of 28 (47% of all ventilated patients) adults intubated for 48 hours or longer, who developed tachypnea (40 ± 9 breaths/min) but whose blood gas exchange met predefined extubation criteria, were evaluated over a 3-month period. INTERVENTIONS: Using a microprocessor-based monitor (Bicore Monitoring Systems Inc, Irvine CA) total patient work of breathing (WOB_{TOT}) was determined by integrating the change in intraesophageal pressure with tidal volume measured with a miniature pneumotachograph positioned at the airway opening. If the patient's WOB_{TOT} was equal to or greater than 0.8 J/L, WOB_{imp} was determined by integrating the changes in carinal pressures with tidal volume. If neither the patient's WOB_{TOT} or WOB_{phys} was excessively greater than that of spontaneous breathing at rest (ie, < 0.8 J/L: normal range, 0.5 to 0.6 J/L), the patient was extubated. MEASUREMENTS & RESULTS: Breathing frequency, peak inspiratory flow rate (PIFR), auto-PEEP ($PEEP_a$), dynamic compliance WOB_{TOT}, WOB_{imp}, resistance to expiratory airway flow (RAW_L) were measured, and WOB_{phys} calculated (WOB_{TOT} minus WOB_{imp}). The means and SDs were calculated, and data were analyzed by unpaired *t* test and linear regression. Six patients (5%) were found to have WOB_{TOT} of < 0.8 J/L and were successfully extubated without determination of WOB_{imp}. Twenty-one patients were found to have an elevated WOB_{TOT} (1.6 ± 0.83 J/L), and had WOB_{imp} measured. In these 21 patients, WOB_{imp} (1.1 ± 0.64 J/L) was twice WOB_{phys} (0.5 ± 0.26 J/L). Extubation was successful in 20 of 21 patients in which WOB_{phys} was determined not to be excessive (ie, < 0.8 J/L). The last patient had an elevated WOB_{phys} (1.4 J/L) and was not extubated until his disease improved later. Overall, reintubation rate was 4%. CONCLUSION: Increased WOB_{TOT} may be misinterpreted as a patient failure (ie, tachypnea) and weaning halted or extubation not done, prolonging intubation. The ability to measure the contribution of WOB_{imp} to WOB_{TOT} can identify those patients who may be safely extubated when WOB_{phys} (WOB_{TOT} or minus WOB_{imp}) is acceptable and the apparent ventilatory insufficiency is related to significant WOB_{imp}.

Noise Measurements during High-Frequency Oscillatory and Conventional Mechanical Ventilation—RJ Berens, CGM Weigle. Chest 1995; 108(4):1026.

OBJECTIVE: To evaluate the noise levels with high-frequency oscillatory ventilation and conventional mechanical ventilation. DESIGN: An observational, prospective study. SETTING: Pediatric intensive care unit. SUBJECTS: The caretakers and environment of the pediatric intensive care unit. INTERVENTIONS: High-frequency oscillatory and conventional mechanical ventilation. MEASUREMENTS & MAIN RESULTS: Caretakers evaluated noise using a visual analog scale. Noise was measured with a decibel meter and an octave band frequency filter. There was twice as much noise perceived by the caretakers and as measured on the decibel A scale. All measures showed significantly greater noise, especially at low frequencies, with high-frequency oscillatory ventilation. CONCLUSIONS: High-frequency oscillatory ventilation exposes the patient to twice as much noise as does the use of conventional mechanical ventilation.

Cardiopulmonary Effects of Positive Pressure Ventilation during Acute Lung Injury—JA Romand, W Shi, MR Pinsky. Chest 1995;108 (4):1041.

STUDY OBJECTIVES: To assess the gas exchange and hemodynamic effects of pressure-limited ventilation (PLV) strategies in acute lung injury (ALI). We hypothesized that in ALI, the reduction of plateau airway pressure (P_{aw}) would be associated with less alveolar overdistention and thus have better hemodynamic and gas exchange characteristics than larger tidal volume (V_T) ventilation. SETTING: Laboratory. DESIGN: Prospective time-controlled sequential animal study. MEASUREMENTS: Right atrial, pulmonary artery, left atrial, arterial, lateral pleural (Ppl), and pericardial (Ppc) pressures, P_{aw}, ventricular stroke volume, mean expired CO_2, and arterial and mixed venous oxygen contents. Airway resistance and static lung compliance were also measured. INTERVENTIONS: Intermittent positive pressure ventilation (IPPV) given before (control) and after induction of ALI by oleic acid infusion (0.1 mL/kg). IPPV at F_{IO_2} of 1, V_T of 12 mL/kg, and frequency adjusted to maintain normocarbia. ALI PLV was given during ALI and defined as that V_T which gave a similar plateau P_{aw} to that of control IPPV. High-frequency jet ventilation (HFJV) and ALI HFJV were also given and defined as frequency within 10% of heart rate and mean P_{aw} similar to that during control IPPV. RESULTS: After ALI, static lung compliance, P_{aO_2}, and pH decreased, whereas airway resistance and P_{aCO_2} increased. For a constant lung volume, Ppl and Ppc were not different between control and ALI. Both absolute dead space (V_D) and intrapulmonary shunt fraction increased after ALI, but absolute V_D was lower with ALI PLV and ALI HFJV when compared with ALI IPPV. Ventilation did not alter hemodynamics during ALI. CONCLUSIONS: Changes in lung volume determine Ppc and Ppl. PLV strategies do not alter hemodynamics but result in less of an increase in V_D/V_T than would be predicted from the obligatory decrease in V_T.

About Egophony—JD Sapira. Chest 1995;108 (3):865.

Egophony is a change in timbre (E*e* to A) but not pitch or volume. It is due to a decrease in the amplitude and an increase in the intensity of the second formant, produced by solid (including compressed lung) interposed between the resonator and the stethoscope head. This explains certain difficulties in learning this valuable but currently neglected sign as well as in understanding certain physiologic false-positive occurrences.

The Effects of Applied vs Auto-PEEP on Local Lung Unit Pressure and Volume in a Four-Unit Lung Model—RM Kacmarek, M Kirmse, M Nishimura, H Mang, WR Kimball. Chest 1995; 108(4):1073.

BACKGROUND. The application of positive end-expiratory pressure (PEEP) and maintenance of increased mean airway pressure (MAP) has been associated with improved oxygenation in adult respiratory distress syndrome. Recently, attention has been directed toward elevating MAP by establishing auto-PEEP when ventilating with an inverse inspiratory to expiratory ratio in opposition to applied PEEP. We theorized that FRC distribution and local lung unit end-expiratory pressure (EEP) would be different when equal levels of PEEP were established by applying PEEP or by producing auto-PEEP. METHODS: Using a 4-chamber lung model with each chamber having a different time constant (TC), we applied equal levels of applied PEEP (I:E ratio 1:3) and auto-PEEP (I:E ratio 3:1) and evaluated local lung unit EEP and end-expiratory lung volume (EELV). RESULTS: During all trials with applied PEEP, local lung unit EEP was equal to applied PEEP, whereas during auto-PEEP local EEP differed ($p < 0.01$). At a tracheal auto-PEEP level of 12.7 cm H_2O, the lung unit with the longest TC (slow lung unit) had an EEP of 15.8 cm H_2O, while the shortest TC unit (fast lung unit) had an EEP of 10.1 cm H_2O ($p < 0.01$). Similarly, local EELVs were more maldistributed with auto-PEEP than with applied PEEP. At a tracheal PEEP level of 12.7 cm H_2O, the EELV increase in the slow lung unit with auto-PEEP was 1,054 mL vs 918 with applied PEEP

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Please send the abstract and cover sheet to:

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($p < 0.01$); whereas, the fast lung unit's EELV increase with auto-PEEP was 142 mL compared with 212 mL with applied PEEP ($p < 0.01$). CONCLUSION: Comparing equal levels of auto-PEEP with applied PEEP, a greater maldistribution of local lung unit EEP and EELV was established with the auto-PEEP. During auto-PEEP, the greatest EEP and EELV occurred in the slow lung unit, and the lowest EEP and EELV developed in the fast lung unit.

Nitric Oxide Administration Using Constant-Flow Ventilation—JW Skimming, S Cassin, PB Blanch. Chest 1995;108(4):1065.

Nitric oxide (NO) gas is known as both a vasodilator and a toxin. It can react with oxygen to form compounds more toxic than itself, such as nitrogen dioxide (NO_2). The reactions are time dependent; thus, infusing NO into breathing circuits as close to ventilated subjects as possible may help minimize toxic byproduct exposure. Unfortunately, flowrates commonly used with mechanical ventilation favor laminar gas flow (streaming) within the breathing circuits. Streaming could delay mixing of NO with other inhaled gases. This mixing delay may interfere with accurate monitoring and/or delivery of NO. We tested the hypothesis that streaming of NO infused by constant flow into the inspiratory limb of a constant flow mechanical ventilation system can lead to NO concentration delivery estimate errors. We then compared the NO_2 concentrations at the ventilator Y-piece with 3 different NO mixing methods: blending the gases before they reach the breathing circuit inspiratory limb, infusing NO directly into the breathing circuit inspiratory limb far enough from the Y-piece to ensure thorough mixing, and infusing NO directly into the breathing circuit inspiratory limb immediately before the gases reach an in-line mixing device placed close to the Y-piece. Our results indicate that streaming can lead to NO concentration delivery estimate errors and that these errors can be characterized by measuring NO concentration variations across the inspiratory tubing's luminal diameter. NO_2 concentrations measured at the ventilator Y-piece were dependent on NO concentrations ($p < 0.0001$), NO delivery methods ($p < 0.0001$), and interactions between NO concentrations and NO delivery methods ($p < 0.0001$). We conclude that gas streaming and toxic byproduct exposure should be considered together when choosing an NO delivery method.

Measurements of Morning and Evening Airflow Grossly Underestimate the Circadian Variability of FEV_1 and Peak Expiratory Flow Rate in Asthma—GE D'Alonzo, VW Steinijans, A Keller. Am J Respir Crit Care Med 1995; 152:1097.

Peak expiratory flow rate (PEF) or forced expiratory volume in 1 s (FEV_1) monitoring is an important clinical tool to assess the degree of 24-hour variation (circadian) in lung function, which correlates with the degree of airway hyperresponsiveness. Ideally, PEF measurements should be taken frequently during the 24-hour period to reflect the true variability. In practice, however, measurements are generally taken twice daily, usually upon arising and in the evening before bedtime. The effect of reducing the frequency of lung function measurement on the reported 24-hour time average (mesor) and the circadian variability were evaluated. Four measurements taken 8 hours apart and 2 measurements derived upon arising and the other, either 12 hours later or before retiring, were compared with the full 24-hour lung function profile, which included 13, 2-hourly measurements. Based on the data from a 24-hour lung function study in 20 adult patients with asthma, the corresponding values were separately compared at baseline and under 2 theophylline treatments, one being administered twice daily and the other once daily. The results are as follows: Whereas the 24-hour time average (mesor) is hardly effected by the reduced measurements schemes, variability was grossly underestimated. Only 60-80% of the actual variability is picked up in the case of the 4, 8-hourly measurements, and only 20-45% in the case of the 2 measurements. The loss of accuracy in assessing 24-hour airflow variability has potentially important implications on clinical practice and the investigation of therapeutics in asthma. In conclusion, it would be ideal to be able to perform airflow measurements every 2 hours around the clock, but this is clinically impractical. While the error in assessing circadian airflow variability is unacceptably high in taking only 2 measurements, it is reduced to acceptable levels by taking 4 measurements a day.

Maternal Smoking during Pregnancy: Effects on Lung Function during the First 18 Months of Life—JB Tager, L Ngo, JP Hanrahan. Am J Respir Crit Care Med 1995;152:977.

The present investigation evaluated the effects of maternal smoking during pregnancy on the growth of lung function during the first 18 months of life in 159 infants who were part of a longitudinal study of the effects of maternal smoking on respiratory health. Infant pulmonary function was assessed at 2-6 weeks of age and at 4-6, 9-12, and 15-18 months of age by partial expiratory flow-volume curves and helium dilution FRC. Maternal smoking was assessed by standard questionnaire and urine cotinine measurements. Maternal smoking during pregnancy was associated with a reduction of 9.4 ± 4.3 mL ($p = 0.029$) for FRC and 33 ± 12.3 mL/s ($p = 0.008$) reduction for flow at FRC ($\dot{V}_{FRC}$) after controlling for the effects of growth (length). The effect of maternal smoking was greater for female infants than for male infants. At 1 year female infants exposed in utero were predicted to have a 16% reduction in $\dot{V}_{FRC}$ compared with 5% for male infants. No sex difference was seen for FRC or $\dot{V}_{FRC}$/FRC. Exposure to environmental tobacco smoke in the postnatal period was not significantly related to reduced FRC or $\dot{V}_{FRC}$. These data provide further evidence that maternal smoking during pregnancy may play a greater role than postnatal and childhood exposure on the observed effects on lung function in children.

Postoperative Physical Therapy after Coronary Artery Bypass Surgery—D Johnson, C Kelm, T To, T Hurst, C Naik, I Gulka, D Thomson, K East, J Osachoff, I Mayers. Am J Respir Crit Care Med 1995;152:953.

Coronary artery bypass surgery is frequently complicated by postoperative atelectasis. Although routinely prescribed, the efficacy of any specific chest physical therapy is not well established. We studied patients at a university center undergoing elective coronary artery bypass surgery. Based upon chest X-ray criteria at extubation, patients (n = 228) were classified as demonstrating greater or lesser degrees of atelectasis. Those with a lesser degree of atelectasis were randomized to receive either early mobilization or sustained maximal inflations (SMI). Those with greater a degree of atelectasis were separately randomized to receive either SMI or single-handed percussions (SSP). We found the extent of atelectasis at extubation did not predict the risk of developing pneumonia. Hospital stays and intensive care unit stays were similar regardless of treatment. Physical therapy costs were highest in the most labor-intensive therapy group (SSP). We conclude that postoperative respiratory dysfunction is common but does not commonly cause significant morbidity or prolong hospital stay. Adding SMI to patients with minimal atelectasis at extubation does not improve clinical outcomes. Similarly, adding SSP to patients with marked atelectasis does not improve outcomes over those obtained with SMI and early ambulation.

Reference Values of Arterial Oxygen Tension in the Middle-Aged and Elderly—I Cervert, MC Zoia, F Fanfulla, L Spagnolatti, L Berrayah, M Grassi, C Tinelli. Am J Respir Crit Care Med 1995;152:934

The lack of available reference values of arterial P_{O_2}, particularly for elderly persons, led us to study a sample of 194 normal nonsmoking subjects, equally distributed over all age ranges from 40 to 90 years. The radial artery was punctured and blood samples were taken and analyzed on an automated, computerized gas-analyzer. The trend of the mean values of P_{aO_2} in the 5-year class intervals of age showed a clear decline up to the 70- to 74-year class, and then an inversion. The 2 regression lines intersecting at this point provided a better fit to the data than did a single regression line ($Ry^2 - R_1^2 = 0.918 - 0.678 = 0.24$; $F = 20.49$, $p = 0.0027$). The relationship of P_{aO_2}

with age was thus subsequently considered for the 2 subgroups (40 to 74 years; ≥ 75 years) identified on the basis of this cutoff. Because of the significant influence on P_{aO_2} of age, body-mass index (BMI), and P_{aCO_2} in the group 40 to 74 years of age, the following reference equation was constructed: P_{aO_2} (mm Hg) = 143.6 − (0.39 · age) − (0.56 · BMI) − (0.57 · P_{aCO_2}); R_2 = 0.28; SEE = 7.48; $p < 0.0001$. For subjects ≥ 75 years old, for whom there was no correlation with age, BMI, or P_{aCO_2}, only the mean ±SD and 5th percentile of P_{aO_2} were reported (83.4 ± 9.15 mm Hg and 68.4 mm Hg, respectively). P_{aCO_2} values were not correlated with either age or BMI; the mean ±SD was 35.79 ± 3.87 mm Hg. Our study yields appropriate reference values of P_{aO_2}, including the lower and upper limits for subjects aged 40 to 90 years, that can be used routinely in pulmonary function laboratories.

Supplemental Oxygen during Sleep in Children with Sleep-Disordered Breathing—CL Marcus, JL Carroll, O Bamford, P Pyzik, GM Loughlin. Am J Respir Crit Care Med 1995;152:1297.

Supplemental O_2 is sometimes used to treat children with the obstructive sleep apnea syndrome (OSAS). However, its effects have not been studied. We therefore evaluated the use of supplemental O_2 during sleep in children with OSAS. Oxygen and room air were delivered via nasal cannula at 1 L/min for 4 hours each in a randomized, double-blind fashion. Twenty-three children were studied (mean age, 5 ± 3 [SD] years). Patients had a higher mean S_{aO_2} and higher S_{aO_2} nadir when breathing O_2. There was no difference in the number (10.9 ± 20.6/hours on O_2 vs 13.5 ± 19.3 on room air) or duration of obstructive apneas. Although there was no overall change in end-tidal P_{CO_2} when patients breathed O_2, 2 children showed a significant increase. We conclude that breathing supplemental O_2 during sleep in children with OSAS results in improved oxygenation and in most cases does not exacerbate sleep-disordered breathing. However, end-tidal P_{CO_2} should be monitored in children with OSAS receiving O_2 therapy. We speculate that supplemental O_2 does not depress the ventilatory drive during sleep in most children with OSAS.

Factors Limiting Exercise Performance in Long-Term Survivors of Bronchopulmonary Dysplasia—P Santuz, E Baraldi, P Zaramella, M Filippone, F Zacchello. Am J Respir Crit Care Med 1995;152:1284.

The long-term impairment of pulmonary function during exercise was assessed in 12 children, aged 6-12 years, who developed BPD after prematurity (gestational age 30 ± 2 weeks [mean ±SD] and birthweight 1,400 ± 335 g) and 16 age-, sex-, and physical activity-matched healthy children born at term, who served as controls. The children performed pulmonary function tests at rest and a maximal stepwise exercise on a treadmill. Oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide output ($\dot{V}_{CO_2}$), and minute ventilation ($\dot{V}_E$) were monitored during the run. Baseline mean spirometric values (% of predicted) were in the normal range for both groups but were lower in BPD children with respect to control children ($p < 0.05$). At rest, arterial oxygen saturation (S_{aO_2}) was ≥ 98% in all BPD children, but at peak exercise, 4 of them had a S_{aO_2} fall ≥ 4%. The postexercise FEV_1 fall, with respect to the baseline, was 8 ± 6% in BPD and 2 ± 1% in control children ($p < 0.01$). Maximum $\dot{V}_{O_2}$ and $\dot{V}_E$ were significantly lower in BPD children with respect to the control group (25.2 ± 10.3 versus 37.1 ± 10.4 mL/min/kg and 20.8 ± 9.4 versus 30.7 ± 7.9 L/min, respectively, both $p < 0.01$). Also, at submaximal levels of exercise dynamic, $\dot{V}_{O_2}$ and $\dot{V}_E$ responses were significantly lower in the BPD group (ANOVA, $p < 0.001$), with a ventilatory pattern characterized by lower tidal volumes. Anaerobic threshold was 20.6 ± 9 in BPD and 28.8 ± 8.6 mL O_2/min/kg in healthy children ($p < 0.05$). Running time was 6.1 ± 1.3 min in BPD and 7.9 ± 2.6 min in control children ($p < 0.05$). No difference was found in maximal heart rate. We conclude that asymptomatic school-aged survivors of BPD show evidence of striking disturbances in ventilatory response to exercise and reduced aerobic capacity compared with healthy children, even if pulmonary function at rest is only slightly impaired.

Albuterol Delivery in a Model of Mechanical Ventilation: Comparison of Metered-Dose Inhaler and Nebulizer Efficiency—P Diot, L Morra, GC Smaldone. Am J Respir Crit Care Med 1995;152:1391.

Using an in vitro model, we compared efficiencies of jet nebulizers and metered-dose inhalers (MDI) with actuator devices to deliver albuterol in various conditions of mechanical ventilation. Factors tested included influence of humidification, MDI actuator device (Aerovent® spacer or Marquest 172275 MDI adaptor), and synchronization of MDI to the respiratory cycle. With the nebulizer (AeroTech II®) filled with 2.5 mg albuterol sulfate in 3 mL water and run until dry, inhaled mass was 42 ± 2.6% and mass median aerodynamic diameter (MMAD) was 1.3 μm on a nonhumidified circuit. With the MDI + Aerovent®, the inhaled mass percentage per 90 μgm puff was 15.4 ± 0.2% with humidification and 25.1 ± 3.7% without humidification, actuations being synchronized with the beginning of inspiration and separated by a 1-minute pause. Failure to synchronize actuations with inspiration or to pause 1 minute resulted in significant reductions in inhaled mass (by 35 and 72%, respectively). The Marquest adaptor was less efficient, with an inhaled mass of 7.2 ± 0.7% under optimal conditions. Metered-dose inhaler actuation during expiration resulted in relatively large particles (MMAD = 2.0 μm). All other MDI actuations led to essentially biphasic distributions, with particles greater than 1 μm following a distribution similar to the nebulizer and the overall MMAD estimated to be 0.22 μm. The AeroTech II® delivered a cumulative 1,000 μg of drug (2,500 × 0.40) over 40 minutes. To achieve that amount, the MDI connected to the Aerovent® and used in its most efficient sequence would require 45 timed puffs (90 μg per puff, 25.1% mean inhaled mass) and take 45 minutes of an experienced therapist's time. We conclude that nebulizers and MDI/spacers can both be effective in delivering aerosolized medications, but drug delivery can be significantly reduced if proper technique in setting up and using both devices is not followed. Physicians and respiratory therapists should be formally trained in the principles of aerosol delivery with mechanical ventilation.

Clinical Risk Factors for Pulmonary Barotrauma: Multivariate Analysis—RB Gammon, MS Shin, RH Groves Jr, JM Hardin, C Hsu, SE Buchalter. Am J Respir Crit Care Med 1995; 152:1235.

Previous investigations have suggested that elevated airway pressures increase the risk of ventilator-induced pneumothorax. However, risk factor analysis using multivariate techniques has not been done. We investigated the hypothesis that airway pressures would not independently correlate with pneumothorax when underlying disease was considered. All ventilated patients over a 1-year period in the Hohenburg Critical Care Unit at the University of Alabama were followed until death or discharge from the ICU. Ventilator data were collected daily and the presence of pneumomediastinum and pneumothorax determined by review of chest radiographs. Maximal values of airway pressures, minute ventilation, tidal volume, and respiratory rate, as well as age, sex, and underlying disease, were entered into logistic regression analysis. A total of 168 patients was studied, and 20 experienced pneumothorax. Multivariate analysis of the entire ventilated population revealed that only the presence of ARDS independently correlated with pneumothorax. A similar analysis performed on the ARDS population revealed independent correlation only with male sex. Trends toward elevation in airway pressures were seen that did not reach statistical significance. We conclude that development of pneumothorax is most closely correlated with underlying disease, specifically ARDS, and that the associations previously noted between airway pressures and barotrauma largely relate to the occurrence of high airway pressures in ARDS.

The Physiologic Effects of Inhaled Amphotericin B—J Dubois, T Bartter, J Gryn, MR Pratter. Chest 1995;108:750.

Our institution used an experimental protocol for the use of inhaled amphotericin B as a prophylactic measure to prevent fungal disease in severe-

ly immunocompromised patients. We did a prospective study of the physiologic effects of amphotericin B administration. We looked specifically at oxygen saturation levels, peak flow values, and symptoms of patients given amphotericin B. We collected data on a series of 18 patients and of 132 amphotericin B administrations. Four (22%) of the patients stopped treatments because of nausea and vomiting which were believed to be due to the inhaled amphotericin B. For the remaining patients, no treatment was stopped because of symptoms or physiologic changes caused by amphotericin B, although there were 9 instances of clinically significant bronchospasm as defined by a drop in peak flow of 20% or more, 9 clinically relevant increases in cough, and 3 clinically relevant increases in dyspnea. Forty-eight percent of the clinically relevant changes occurred in patient 8. Another 16% occurred in asthmatic subjects who were significantly more likely ($p = 0.03$) to experience a 20% or more drop in peak flow than were patients without asthma. The physiologic profile of the response to inhaled amphotericin B is acceptable.

Liquid Ventilation in Adults, Children, and Full-Term Neonates—RB Hirschl, T Pranikoff, P Gauger, RJ Schreiner, R Dechert, RH Bartlett. Lancet 1995;346:1201.

We evaluated the safety and efficacy of partial liquid ventilation in a series of 19 adults, children, and neonates who were in respiratory failure and on extracorporeal life support. During partial liquid ventilation, the alveolar-arterial oxygen difference decreased from 590 (SE 25) to 471 (42) mm Hg ($p = 0.0002$) and static pulmonary compliance increased from 0.18 (0.04) to 0.29 (0.04) $mL \cdot cm\ H_2O^{-1} \cdot kg^{-1}$ ($p = 0.0002$). Eleven patients (58%) survived. These preliminary data suggest that partial liquid ventilation can be safely used in patients with severe respiratory failure and may improve lung function.

Menopause, Postmenopausal Estrogen Preparations, and the Risk of Adult-Onset Asthma: A Prospective Cohort Study—RJ Troisi, FE Speizer, WC Willett, D Trichopoulos, B Rosner. Am J Respir Crit Care Med 1995;152:1183.

We prospectively evaluated the association of hormone replacement therapy and asthma incidence in a cohort of pre- and postmenopausal women 34-68 years of age. During 582,135 person-years of follow-up between 1980 and 1990, 726 new cases of asthma were documented. Postmenopausal women who were never users of replacement hormones had a significantly lower age-adjusted risk of asthma than premenopausal women (relative risk = 0.65; 95% confidence interval [CI] = 0.46 to 0.92). Among naturally menopausal women, the age-adjusted relative risk of asthma for ever use of postmenopausal hormones was 1.49 (95% CI = 1.10 to 2.00); for current use of hormones (conjugated estrogens with or without progesterone), 1.50 (95% CI = 0.98 to 2.30); and for past use, 1.52 (95% CI = 1.08 to 2.13), compared with never use of hormones. Ever users of 10 or more years' duration had twice the age-adjusted risk of asthma compared with women who never used postmenopausal hormones (95% CI = 1.39 to 2.87). Among current users of conjugated estrogens, there was a positive dose-response demonstrated between daily dose and asthma risk (p for trend = 0.007). While confirmatory studies are warranted, these data suggest that estrogen plays a role in the pathophysiology of asthma and that long-term use and/or high doses of postmenopausal hormone therapy increase subsequent risk of asthma.

The Safety of Air Transportation of Patients with Advanced Lung Disease: Experience with 21 Patients Requiring Lung Transplantation or Pulmonary Thromboendarterectomy—MR Kramer, DJ Jakobson, C Springer, Y Donchin. Chest 1995;108:1292.

Air travel can cause severe respiratory decompensation in a patient with advanced lung disease due to high altitude hypoxemia. We report our experience in flying 21 patients with advanced lung disease to a medical center remote from Israel for lung transplantation or pulmonary thromboendarterectomy (PTE). All patients had severe lung disease with marked hypoxemia (P_{aO_2}, 40 to 59) and 16 had significant pulmonary hypertension. Nine patients (with emphysema and pulmonary fibrosis) required single lung transplant, 4 (with cystic fibrosis and emphysema) required double-lung transplant, 6 (with primary or secondary pulmonary hypertension) required heart-lung transplant, and 2 (with major vessel pulmonary thrombosis) required PTE. All patients were flown by commercial aircraft to centers located 2,634 to 13,181 km away from Israel. Length of flight was between 4 and 21 hours. Patients were given oxygen supplementation during the flight and were monitored by portable oximeters. All but 3 patients were hemodynamically stable and 19 of them were escorted by physicians. All but one hemodynamically unstable patient who died on board arrived safely at their destinations. We conclude that with careful preparation, sufficient oxygen supply, oximetric monitoring, and medical escort, almost any patient with severe lung disease can travel by air to any necessary destination.

Spirometry and Maximal Respiratory Pressure References from Healthy Minnesota 65- to 85-Year-Old Women and Men—PL Enright, AB Adams, PJR Boyle, DL Sherrill. Chest 1995; 108(3):663.

OBJECTIVE: To obtain spirometry and maximal respiratory pressure (MRP) reference values for elderly persons. DESIGN: Survey. SETTING: General community. PARTICIPANTS: Four hundred seventy-one healthy ambulatory white women and men age 65 + years. METHODS: A stringent spirometry quality assurance program exceeded American Thoracic Society recommendations. A "healthy" subgroup of 176 women and 112 men between the ages of 65 and 85 years were identified by excluding those with conditions that negatively influenced FEV_1 in a multiple regression analysis. Reference equations and normal ranges for FEV_1, FVC, FEF25-75%, peak flow, and maximal inspiratory and expiratory pressures (MRPs) were determined from the healthy group with good quality maneuvers. RESULTS: Less than 10% of the subjects were unable to perform 3 acceptable spirometry maneuvers and 10 MRP maneuvers. When the age and height corrected FEV_1s from this group were compared with other spirometry reference studies, mean values from the women were nearly identical to those from Morris, while these men had substantially lower FEV_1 values (by 0.3 to 0.5 L) than elderly men in Crapo's study. Mean peak flow was over 20% higher when compared with previous studies, suggesting greater initial expiratory effort by our subjects. The maximal inspiratory pressure (MIP) values were about 20% higher than those reported by the Cardiovascular Health Study, perhaps because 5 MIP maneuvers were always performed. CONCLUSION: Spirometry and MRP reference values used for elderly patients should come from population studies using similar techniques and with large numbers of subjects over age 65 years. *See the related editorial:* ***The Quest for Normal Values****—JR Rodarte. Chest 1995;108(3):594-595.*

Inability of Clinical History to Distinguish Primary Snoring from Obstructive Sleep Apnea Syndrome in Children—JL Carroll, SA McColley, CL Marcus, S Curtis, GM Loughlin. Chest 1995;108(3):610.

OBJECTIVE: To determine whether primary snoring (PS) could be distinguished from childhood obstructive sleep apnea syndrome (OSAS) by clinical history. DESIGN: Retrospective study of clinical history of 83 children with snoring and/or sleep disordered breathing who were referred for polysomnography. SETTING: Tertiary referral center; pediatric pulmonary sleep apnea clinic. MEASUREMENTS: We evaluated the ability of a clinical obstructive sleep apnea (OSA) score and other questions about sleep, breathing, and daytime symptoms to distinguish PS from OSAS in children. Parents were asked about the child's snoring, difficulty breathing, observed apnea, cyanosis, struggling to breathe, shaking the child to "make him or her breathe," watching the child sleep, afraid of apnea, the frequency and loudness of snoring, and daytime symptoms such as excessive daytime sleepiness (EDS). RESULTS: Based on polysomnography results, 48 patients were classified as PS and 35 as OSAS. Peak end-tidal CO_2 (49 ± 3.2 vs 55 ± 8.2 [SD] mm Hg); lowest arterial oxygen saturation measured by pulse oxime-

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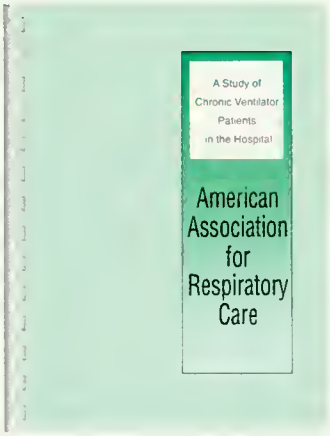

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try (95 ± 1.9 vs 82 ± 14%); and apnea/hypopnea index (0.27 ± 0.3 vs 8.4 ± 6 events/hours) indicated that the diagnostic criteria for PS versus OSA were reasonable. There were no differences between PS and OSA patients with respect to age, sex, race, failure to thrive, obesity, history of EDS, snoring history, history of cyanosis during sleep, or daytime symptoms except for mouth breathing. There were no significant differences in sleep variables between PS patients and those with any severity of OSAS. The OSA score misclassified about 1 of 4 patients. Comparing PS and OSA patients, significant findings were daytime mouth breathing (61 vs 85%; p = 0.024); observed apnea (46 vs 74%; p = 0.013); shaking the child (31 vs 60%; p = 0.01); struggling to breathe (58 vs 89%; p = 0.003); and afraid of apnea (71 vs 91%; p = 0.028). However, none of these were sufficiently discriminatory to predict OSAS. CONCLUSION: We conclude that PS in children cannot be reliably distinguished from OSAS by clinical history alone.

Evaluation of Carbon Dioxide Rebreathing during Pressure Support Ventilation with Airway Management System (BiPAP) Devices— F Lofaso, L Brochard, D Touchard, T Hang, A Harf, D Isabey. Chest 1995;108:772.

The purpose of this study was to evaluate whether carbon dioxide (CO_2) rebreathing occurs in acute respiratory failure patients ventilated using the standard airway management system (BiPAP pressure support ventilator; Respironics; Murrysville PA) with positive inspiratory airway pressure and a minimal level of positive end-expiratory pressure (PEEP) and whether any CO_2 rebreathing may be efficiently prevented by the addition of a nonrebreathing valve to the BiPAP system circuit. In the first part of the study, the standard device was tested on a lung model with a nonrebreathing valve (BiPAP-NRV) and with the usual Whisper Swivel connector (BiPAP-uc). With the BiPAP-uc device, the resident volume of expired air in the inspiratory circuit at the end of expiration (RVEA) was 55% of the tidal volume (V_T) when the inspiratory pressure was 10 cm H_2O and the frequency was at 15 cycles per minute. The BiPAP-NRV device efficiently prevented CO_2 rebreathing but resulted in a slight decrease in V_T, which was due to a significant increase in external PEEP (2.4 vs 1.3 cm H_2O) caused by the additional expiratory valve resistance. For similar reasons, both the pressure swing necessary to trigger pressure support and the imposed expiratory work were increased in the lung model when the nonrebreathing valve was used. In the second part of the study, 7 patients weaned from mechanical ventilation were investigated using a randomized crossover design to compare 3 situations: pressure support ventilation with a conventional intensive care ventilator (CIPS), BiPAP system use, and BiPAP-NRV. When we compared the BiPAP system use with the other 2 systems, we observed no significant effect on blood gases but found significant increases in V_T, minute ventilation, and work of breathing. These findings are experimental and are clinical evidence that significant CO_2 rebreathing occurs with the standard BiPAP system. This drawback can be overcome by using a nonrebreathing valve, but only at the expense of greater expiratory resistance.

Volume-Controlled Inverse Ratio Ventilation in Oleic Acid Induced Lung Injury: Effects on Gas Exchange, Hemodynamics, and Computed Tomographic Lung Density— U Ludwigs, C Klingstedt, S Baehrendtz, G Wegenius, G Hedenstierna. Chest 1995;108:804.

OBJECTIVE: To compare volume-controlled inverse ratio ventilation (VCIRV) with volume-controlled ventilation with conventional inspiratory to expiratory (I:E) ratio (VCV PEEP) at equal levels of end-expiratory pressure. DESIGN: Animal study using an oleic acid lung injury model with random application of VCV PEEP and VCIRV. SETTING: Experimental investigation at the Department of Clinical Physiology at Uppsala University. ANIMALS: Seven pigs. INTERVENTIONS: VCV PEEP, VCIRV at an end-expiratory pressure level of 10 cm H_2O. MEASUREMENTS & RESULTS: Lung mechanics, hemodynamics, gas exchange, and functional residual capacity. Recruitment of lung tissue, regional lung density, and distribution of inspired gas by computed tomography. Mean and peak airway pressures were 22 ± 4 and 41 ± 8 cm H_2O with VCIRV and 18 ± 2 and 45 ± 7 cm H_2O with VCV PEEP. Cardiac output and arterial oxygen tension were equal with VCV PEEP and VCIRV as were static compliance, physiologic dead space, and functional residual capacity. End-expiratory, end-inspiratory, and CT densities during a full ventilatory cycle were not statistically different and the amounts of nonaerated and poorly aerated lung areas were of equal size with VCV PEEP and VCIRV. CONCLUSIONS: VCIRV was comparable to VCV PEEP at similar PEEP levels in alveolar recruitment, aeration of the lung tissues, and in oxygenating the blood. Since cardiac output also remained unchanged, oxygen delivery to peripheral tissues did not differ significantly between the 2 modes. Neither method has thus proved superior to the other one.

Effect of Routine Emergency Department Triage Pulse Oximetry Screening on Medical Management—WR Mower, C Sachs, EL Nicklin, P Safa, LJ Baraff. Chest 1995;108(5):1297.

PURPOSE: To determine the utility of routine triage pulse oximetry screening in emergency department (ED) patients. DESIGN: Prospective study using pulse oximetry to measure oxygen saturation of ED patients at triage. Saturation values were disclosed to physicians only after they completed medical evaluations and were ready to discharge or admit each patient. We measured changes in medical management initiated after disclosure of pulse oximetry values. SETTING & PARTICIPANTS: The study included 14,059 consecutive patients presenting to triage at a university ED. MEASUREMENTS: Changes in select diagnostic tests: chest radiography, CBC count, spirometry, arterial blood gases, pulse oximetry, and ventilation-perfusion scans; treatments: antibiotics, β-agonists, supplemental oxygen; and hospital admission and final diagnoses that occurred after disclosure of triage pulse oximetry values. RESULTS: Of 1,175 patients having triage pulse oximetry values less than 95%, physicians ordered repeat pulse oximetry on 159 (13.5%), additional chest radiography on 5.4%, CBC count on 3.1%, arterial blood gases on 2.9%, spirometry on 0.9%, and ventilation-perfusion scans on 0.3%. Physicians ordered 178 new therapies on 134 patients (11.4%), including supplemental oxygen for 6.5%, antibiotics for 3.9%, and β-agonists for 1.8%. Thirty-five patients (3.0%) initially scheduled for hospital discharge were subsequently admitted. Physicians changed or added diagnoses in 77 patients (6.6%). CONCLUSIONS: Providing physicians with routine triage pulse oximetry measurements resulted in significant changes in medical treatment of these patients.

Varying Nicotine Patch Dose and Type of Smoking Cessation Counseling— DE Jorenby, SS Smith, MC Fiore, RD Hurt, KP Offord, IT Croghan, JT Hays, SF Lewis, TB Baker. JAMA 1995;274(17):1347.

OBJECTIVE: To compare the efficacy and safety of 22-mg and 44-mg doses of transdermal nicotine therapy when it is paired with minimal, individual, or group counseling to improve smoking cessation rates. DESIGN: An 8-week clinical trial (4 weeks double-blind followed by 4 weeks open label) using random assignment of participants to both dose (22 or 44 mg) and counseling (minimal, individual, or group) conditions. PARTICIPANTS: Daily cigarette smokers (≥ 15 cigarettes per day for at least 1 year) who volunteered to participate in a study of smoking cessation treatment. A total of 504 participants were enrolled at 2 sites. INTERVENTION: Four weeks of 22- or 44-mg transdermal nicotine therapy followed by 4 weeks of dosage reduction (2 weeks of 22 mg followed by 2 weeks of 11 mg). Counseling consisted of a self-help pamphlet (minimal); a self-help pamphlet, a brief physician motivational message, and 3 brief (< 15 minutes) follow-up visits with a nurse (individual); or the pamphlet, the motivational message, and 8 weekly 1-hour group smoking cessation counseling visits (group). All participants returned weekly to turn in questionnaires and for assessment of their smoking status. MAIN OUTCOME MEASURES: Abstinence from smoking was based on self-report, confirmed by an expired carbon monoxide concentration lower than 10 ppm. Withdrawal severity was assessed by means

of an 8-item self-report questionnaire completed daily. RESULTS: Smoking cessation rates for the 2 nicotine patch doses and 3 levels of counseling did not differ significantly at either 8 weeks or 26 weeks following the quit date. Among those receiving minimal contact, the 44-mg dose produced greater abstinence at 4 weeks than did the 22-mg dose (68% vs 45%; $p < 0.01$). Participants receiving minimal-contact adjuvant treatment were less likely to be abstinent at the end of 4 weeks than those receiving individual or group counseling (56% vs 67%; $p < 0.05$). The 44-mg dose decreased desire to smoke more than the 22-mg dose, but this effect was not related to success in quitting smoking. Transdermal nicotine therapy at doses of 44 mg produced a significantly greater frequency of nausea (28%), vomiting (10%), and erythema with edema at the patch site (30%) than did a 22-mg dose (10%, 2%, and 13%, respectively; $p < 0.01$ for each adverse effect). Three serious adverse events occurred during use of the 44-mg patch dose. CONCLUSIONS: There does not appear to be any general, sustained benefit of initiating transdermal nicotine therapy with a 44-mg patch dose or of providing intense adjuvant smoking cessation treatment. The 2 doses and all adjuvant treatments produced equivalent effects at the 26-week follow-up, and the higher patch dose produced more adverse effects. Higher-dose (44-mg) nicotine replacement does not appear to be indicated for general clinical populations, although it may provide short-term benefit to some smokers attempting to quit with minimal adjuvant treatment. *See the related editorial:* ***Treatment of Nicotine Dependence—Is More Better?****—JR Hughes. JAMA 1995;274(17):1390-1391.*

High-Dose Nicotine Patch Therapy: Percentage of Replacement and Smoking Cessation—LC Dale, RD Hurt, KP Offord, GM Lawson, IT Croghan, DR Schroeder. JAMA 1995;274(17):1353.

OBJECTIVE: To assess the level of nicotine replacement, evidence of nicotine toxicity, and withdrawal symptom relief with placebo and 11-, 22-, and 44-mg/d doses of transdermal nicotine. A secondary objective was to assess short- and long-term smoking cessation rates. DESIGN: Randomized, double-blind, placebo-controlled inpatient/outpatient trial. SUBJECTS: Seventy-one cigarette smokers stratified according to light ($n = 23$), moderate ($n = 24$), and heavy ($n = 24$) smoking rates. INTERVENTIONS: After baseline measures were obtained, subjects were randomly assigned to placebo or an 11 -, 22-, or 44-mg/d dose of transdermal nicotine and admitted to a special hospital unit for intensive inpatient treatment of nicotine dependence. During the 6-day inpatient stay, daily nicotine and cotinine levels were determined from trough and peak blood samples. Outpatient patch therapy continued for 7 weeks following the hospital stay, and those initially assigned to placebo were randomly assigned to 11 or 22 mg/d. At week 4, the dosage of those initially assigned to 44 mg/d was reduced to 22 mg/d. MAIN OUTCOME MEASURES: Percentage of replacement of cotinine was calculated by dividing the steady-state levels attained during patch therapy by the corresponding baseline levels. Abstinence from smoking was verified by expired air carbon monoxide. Withdrawal symptoms and nicotine toxicity were assessed daily through questionnaires during the inpatient stay. Follow-up visits were at 3, 6, 9, and 12 months. RESULTS: There was a statistically significant relationship between baseline smoking rate and baseline trough and peak blood cotinine levels ($r_s = 0.39$, $r_s = 0.45$; $p < 0.001$ in both instances). A dose-response relationship was observed with higher patch doses, which produced a higher percentage of cotinine replacement and better withdrawal symptom relief. Only one subject (a light smoker assigned to the 44-mg dose) developed signs of nicotine toxicity. There was a positive association between the week 2 patch dose and the biochemically confirmed abstinence at the end of patch therapy ($p = 0.007$) but not for subsequent follow-up times. A higher percentage of cotinine replacement was associated with the higher 8-week smoking abstinence rate ($p = 0.03$), an association not found at long-term follow-up. CONCLUSION: A 44-mg/d dose of nicotine patch therapy appears to be safe for use in heavy smokers. Cigarette smoking rates can be used to estimate the initial nicotine patch dose. Monitoring blood cotinine levels at baseline and steady state can be used for assessing the adequacy of nicotine replacement. Withdrawal symptom relief can be improved with more complete nicotine replacement. Achieving a greater percentage of nicotine replacement may increase the efficacy of nicotine patch therapy. *See the related editorial:* ***Treatment of Nicotine Dependence—Is More Better?****—JR Hughes. JAMA 1995;274(17):1390-1391.*

Prospective Validation of an Acute Respiratory Distress Syndrome Predictive Score—JE Heffner, LK Brown, CA Barbieri, KS Harpel, J DeLeo. Am J Respir Crit Care Med 1995; 152:1518.

We derived an Acute Respiratory Distress Syndrome Score (ARDS Score) from previously described training set data. To validate its diagnostic accuracy for identifying a complicated course (early death or prolonged intubation) in acute lung injury, 50 patients were prospectively scored using an ARDS Score decision threshold of ≥ 2.5 to discriminate between an uncomplicated (successful extubation after ≤ 14 days) and complicated course. Predictor factors incorporated in the ARDS Score were collected on Day 4 and Day 7 of ARDS and included P_{aO_2}/P_{AO_2} ratio, required positive end-expiratory pressure (PEEP), and chest radiograph progression. The diagnostic accuracy of the ARDS Score for determining a complicated course as well as overall survival was compared with 3 other available indices. Using receiver operating characteristic (ROC) analysis, the ARDS Score and Lung Injury Score (LIS) had the greatest diagnostic accuracy for determining a complicated course, but the Simplified Acute Physiology Score (SAPS Score) (score ≥ 14) more accurately identified survival. The LIS components of static respiratory system compliance (C_{rs}) and chest radiograph description did not differ between patient groups. The interobserver concordance of the dynamic chest radiograph interpretation included in the ARDS Score was significant ($p < 0.05$). We conclude that the previously derived ARDS Score has valid diagnostic accuracy for identifying patients with ARDS who will follow a complicated course.

Arterial Oxygen Saturation in Tibetan and Han Infants Born in Lhasa, Tibet—S Niermeyer, P Yang, Shanmina, Drolkar, J Zhuang, LG Moore. N Engl J Med 1995;333:1248.

BACKGROUND: Reduced oxygen availability at high altitude is associated with increased neonatal and infant mortality. We hypothesized that native Tibetan infants, whose ancestors have inhabited the Himalayan Plateau for approximately 25,000 years, are better able to maintain adequate oxygenation at high altitude than Han infants, whose ancestors moved to Tibet from lowland areas of China after the Chinese military entered Tibet in 1951. METHODS: We compared arterial oxygen saturation, signs of hypoxemia, and other indexes of neonatal well-being at birth and during the first 4 months of life in 15 Tibetan infants and 15 Han infants at 3,658 miles above sea level in Lhasa, Tibet. The Han mothers had migrated from lowland China about 2 years previously. A pulse oximeter was placed on each infant's foot to provide measurements of arterial oxygen saturation distal to the ductus arteriosus. RESULTS: The 2 groups had similar gestational ages (about 38.9 weeks) and Apgar scores. The Han infants had lower birthweights (mean [±SE], 2,773 ± 92 g) than the Tibetan infants (3,067 ± 107 g), higher concentrations of cord-blood hemoglobin (18.6 ± 0.8 g per deciliter, vs 16.7 ± 0.4 in the Tibetans), and higher hematocrit values (58.5 ± 2.4%, vs 51.4 ± 1.2% in the Tibetans). In both groups, arterial oxygen saturation was highest in the first 2 days after birth and was lower when the infants were asleep than when they were awake. Oxygen saturation values were lower in the Han than in the Tibetan infants at all times and under all conditions during all activities. The values declined in the Han infants from 92 ± 3% while they were awake and 90 ± 5% during quiet sleep at birth to 85 ± 4% while awake and 76 ± 5% during quiet sleep at 4 months of age. In the Tibetan infants, oxygen saturation values averaged 94 ± 2% while they were awake and 94 ± 3% during quiet sleep at birth and 88 ± 2% while awake and 86 ± 5% during quiet sleep at 4 months. Han infants had clinical signs of hypoxemia—such as

cyanosis during sleep and while feeding—more frequently than Tibetans. CONCLUSIONS: In Lhasa, Tibet, we found that Tibetan newborns had higher arterial oxygen saturation at birth and during the first 4 months of life than Han newborns. Genetic adaptations may permit adequate oxygenation and confer resistance to the syndrome of pulmonary hypertension and right-heart failure (subacute infantile mountain sickness).

Cognitive Function and Assessment of Lung Function in the Elderly—N Carvalhaes-Neto, H Lorino, C Gallinari, S Escolano, A Mallet, F Zerah, A Harf, I Macquin-Mavier. Am J Respir Crit Care Med 1995;152:1611.

The feasibility of spirometry or respiratory impedance measurements for assessing lung function in the elderly was compared in 208 institutionalized patients with various degrees of cognitive function impairment. Respiratory impedance was determined by the forced oscillation technique. Cognitive function was assessed by the score for the mini-mental state (MMS) examination. Of the 208 patients, 126 had severe cognitive impairment (MMS ≤ 17), 36 had mild impairment (18 ≤ MMS ≤ 23), and 46 had no impairment (MMS ≥ 24). Of the 208 patients, respiratory impedance measurements were possible in 159, whereas in only 85 was spirometry possible. The overall difference between the feasibility rates for the spirometric and respiratory impedance measurements was highly significant ($\chi^2 = 71.4$; $p < 10^{-6}$). The difference between the feasibility rates for the 2 techniques was higher in the group of subjects with severe cognitive impairment than in the groups with mild impairment and no impairment, respectively. Among the 84 patients able to complete both tests, significant correlations were found between the spirometric and respiratory impedance measurements. These results indicate that respiratory impedance measurement seems a more useful tool than spirometry for assessing lung function in elderly patients whenever cognitive function is impaired.

Exertional Oxygen of Limited Benefit in Patients with Chronic Obstructive Pulmonary Disease and Mild Hypoxemia—CF McDonald, CM Blyth, MD Lazarus, I Marschner, CE Barter. Am J Respir Crit Care Med 1995;152:1616.

It is unclear whether short-term benefits from supplemental oxygen translate into improved quality of life in patients with severe COPD. In a 12-week double-blind randomized crossover study, we assessed the effects of supplemental air and oxygen on exercise performance (step tests and 6-minute walking distance [6MWD]) initially and after 2, 6-week periods at home using exertional cylinder air or oxygen. We measured quality of life at baseline and after the 2, 6-week domiciliary periods. The 26 patients (24 males) had a mean age of 73 ± 6 years; mean FEV_1, 0.9 ± 0.4 L; mean D_{LCO}, 10.6 ± 2.4 mL/min/mm Hg; mean resting P_{O_2}, 69 ± 8.5 (range 58 to 82) mm Hg; mean P_{CO_2}, 41 ± 3.3 mm Hg; and mean resting S_{aO_2}, 94 ± 2.1 (mean ± SD). Laboratory tests were performed breathing intranasal air or oxygen at 4 L/min, and measurements were made of S_{aO_2} and Borg dyspnea scores. Supplemental oxygen increased 6MWD and steps by small, statistically significant increments acutely at baseline and after 6 and 12 weeks, without corresponding falls in Borg score. Degree of desaturation at baseline did not correlate with increase in 6MWD or steps achieved at baseline or at 6 or 12 weeks, nor with the domiciliary gas used. There was no difference in 6MWD or steps achieved while breathing supplemental oxygen after 6 weeks of domiciliary oxygen compared with domiciliary air. Small improvements in quality of life indices were found after domiciliary oxygen, and mastery also improved after domiciliary air. There were no differences in quality of life, however, when domiciliary oxygen was compared with domiciliary air. Although oxygen supplementation induced small acute increments in laboratory exercise performance, such improvements had little impact on the patients' daily lives.

Changing Heat and Moisture Exchangers Every 48 Hours Rather than 24 Hours Does Not Affect Their Efficacy and the Incidence of Nosocomial Pneumonia—K Djedaini, M Billiard, L Mier, G Le Bourdelles, P Brun, P Markowicz, P Estagnasie, F Coste, Y Boussougant, D Dreyfuss. Am J Respir Crit Care Med 1995;152:1562.

Heat and moisture exchangers (HME) (Dar-Hygrobac II, Peters) can safely be used every 24 hours for long-term mechanical ventilation and provide a cost-saving alternative to heated humidifiers. We have prospectively determined whether changing HMEs every 48 hours only affects their clinical and bacteriological efficiency in a series of consecutive nonselected ICU patients requiring long-term mechanical ventilation. Two consecutive periods were compared. During period 1, HMEs were replaced every day; during period 2, they were changed every 48 hours. Patients from the 2 periods were similar in terms of age and indication for and overall duration of MV (10 ± 8.6 vs 10 ± 9 days, p = 0.9). Minute ventilation and maximum values for peak airway pressure were identical during the 2 periods. These values were also identical after 1 and 2 days of HME use during period 2, indicating that HME resistance was not increased by prolonged use. Obstruction of the tracheal tube occurred only once in a period 1 patient. The results of quantitative cultures indicate that the maximum and mean levels of bacterial colonization during the 2 periods were similar for the pharynx, trachea, Y-connector, patient, and ventilator side of the HME. More importantly, the incidence of nosocomial pneumonia was similar during the 2 periods (6/61 versus 8/68, p = 0.7). Thus, prolonged HME use is safe and provides a substantial reduction in the cost of mechanical ventilation.

Contribution of the Respiratory Muscles to the Lactic Acidosis of Heavy Exercise in COPD—MPKJ Engelen, R Casaburi, R Rucker, E Carithers. Chest 1995;108:1246.

Patients with COPD usually are limited in their exercise tolerance by a limited ventilatory capacity. Lactic acidosis induced by exercise increases the stress on the ventilatory system due to CO_2 generated by bicarbonate buffering and hydrogen ion stimulation. Patients with COPD are often observed to increase blood lactate levels at low levels of exercise. We wished to determine whether patients with COPD who experience lactic acidosis do so because of respiratory muscle production of lactate. Eight patients with moderate to severe COPD (FEV_1 = 43.5 ± 11.6% predicted) and 5 healthy subjects performed 10 minutes of moderate constant work rate exercise either breathing spontaneously or volitionally increasing their ventilation for 5 minutes to approximate the peak minute ventilation seen during incremental exercise. During volitional increased ventilation, 3% CO_2 was added to the inspirate to prevent alkalosis and hypocapnia. In neither the healthy subjects nor the COPD group was the end-exercise lactate level significantly higher during volitional ventilation increase than during spontaneous ventilation. Further, in the COPD patients, the blood lactate levels during volitional ventilation increase were much lower than during maximal exercise (averaging 2.4 vs 5.3 mmol/L) despite similar ventilation levels (averaging 50 and 53 L/min). We conclude that it is unlikely that the respiratory muscles have an important influence on the blood lactate level elevation seen during maximal exercise in COPD patients.

RESPIRATORY CARE—Who Needs It? A Personal View

David J Pierson MD

Introduction

With this issue RESPIRATORY CARE celebrates 40 years of publication—and, there is a lot to celebrate. I would like to explain why I think this is true, based on my personal observations as a reader of and contributor to the Journal for more than half of those 40 years.

What Does This Have To Do with Me?

My own involvement with RESPIRATORY CARE began in the early 1970s, when as a pulmonary fellow I was introduced to Phil Kittredge at an American College of Chest Physicians meeting by Tom Petty, my mentor. Shortly thereafter I submitted a review I had written on respiratory stimulants, and this was published in 1973. At the conclusion of my fellowship and after a 2-year overseas detour in the Army, I was recruited by Len Hudson to join him at Harborview Medical Center in Seattle and become Medical Director of Respiratory Therapy—a position I still hold nearly 20 years later. I joined the AARC (then the AART) and became a loyal reader of its Journal.

In 1980 RESPIRATORY CARE published a case report that I thought was full of holes. Somewhat arrogantly for a junior faculty member with a very slim résumé, I wrote a letter to Phil Kittredge complaining about it, and listed the documentation and teaching points I thought a good case report should contain. To my surprise he wrote back asking me to expand my points into an editorial on the subject, and in addition asked whether I would like to become Case Reports Editor for the Journal and a member of its Editorial Board. Thus, with a bang, began a 14-year stint on the Editorial Board, something that profoundly influenced the course of my own career and, I hope, had some effect on the Journal and its readers. Along the way I have had the good fortune to guest edit or co-edit 12 individual issues and to contribute more than 100 items to its pages, including research articles, OPEN FORUM abstracts, reviews, editorials, book reviews—and even a poem![1]

The ups and downs of all this, along with my experiences with other publications and with the science and practice of respiratory care in this country and abroad, have enabled me to draw some conclusions about this Journal's contributions and importance to the profession. My observations fall into three general categories: things in which we should take pride, an area of disappointment, and things for which I am personally grateful.

What Has the Journal Accomplished?

It has established the respiratory care profession as a science discipline. This would not have occurred without the Journal. In the absence of a dedicated, scientific journal to serve the profession, papers by respiratory care practitioners and papers reporting research on technical and practical aspects of respiratory care by investigators of all credentials would have been published in other journals that include respiratory care among their various subject areas—at least, those papers among them that managed to gain acceptance by those journals. Respiratory care as a distinct subject of research and clinical application might well not have emerged, and respiratory care practitioners today might still be technicians simply carrying out others' instructions, taking part in practice areas they played no role in shaping.

A person seeking evidence for a high level of science in today's RESPIRATORY CARE need look no further than its OPEN FORUM. The first OPEN FORUM I attended was in Dallas at the 1980 Annual Meeting. It took place in an out-of-the-way ground floor room in which there tended to be about 6 people at any one time—the person at the podium, one or two other people from the presenter's home institution, the same representation for the next scheduled abstract, and Phil Kittredge. Today the OPEN FORUM attracts hundreds of abstract submissions from around the world, has an acceptance rate the same as (or a bit lower than) those of the major medical

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specialty societies, and attracts overflow crowds to several large rooms for simultaneous sessions throughout the duration of the convention. The tremendous growth and outstanding quality of the OPEN FORUM of the 1990s are owed primarily to the tireless efforts of Pat Brougher, now assisted in this effort by Associate Editor, Kaye Weber.

Although not every member of the profession always behaves as a professional and not all respiratory care can be classified as scientific, there is no question that the work that appears in the Journal solidly meets the definitions of both.[2] The steadily increasing professional credibility of the Journal can be tracked by the proportion of doctorate-level RCPs serving on its Editorial Board—currently 80%—and authoring original research papers in its pages. Another strong indicator of the Journal's increasing scientific stature is the rigor of its peer review process for submitted manuscripts.

It has provided the health-care community with a unique source of useful clinical information. In addition to publishing important original research, RESPIRATORY CARE provides clinicians with practical reviews and other articles addressing topics that are relevant to their daily practice, but about which there is often a dearth of available literature. These articles are often unique, providing background information and practical help not to be found in any other journal. The main sources for such features, both new since 1980, have been the annual RESPIRATORY CARE Journal conferences and the papers developed from the New Horizons in Respiratory Care Symposia at the AARC Annual Meeting.

The 13 journal conferences to date have produced 25 issues of the Journal, each focused on an important area of clinical practice, and each containing discussions of topics directly pertinent to the clinician's everyday activities. Ten "New Horizons" symposia have yielded 68 articles and 12 accompanying editorials.[3] In both series the authors of individual articles have been charged with explaining the importance of their topics and offering practical advice to the reader on the basis of their experience and expertise, even when applicable research is incomplete or even completely lacking. The author list for these features reads like a "Who's Who" of respiratory care and pulmonary medicine of the last 20 years, and includes virtually all of the main 'players' in the subject areas discussed.

It has established standards of care for the profession. In the last several years the Journal has become the most important source for published guidelines and practice standards in the broad field of respiratory care. The AARC's Clinical Practice Guidelines, all published in RESPIRATORY CARE, are exerting an important influence on public policy and reimbursement in this country, and are rapidly becoming incorporated into institutional policy and procedure manuals around the world. In addition, the Journal's consensus conference issues on aerosol therapy, the essentials of mechanical ventilation, and the assessment of innovation in mechanical ventilation, have established benchmarks that will continue to improve patient care through their influence on manufacturers, regulators, and policy makers as well as clinicians.

It has attained the largest circulation of any journal in the field. RESPIRATORY CARE's monthly run of nearly 40,000 copies exceeds those of the *American Journal of Respiratory and Critical Care Medicine*, *Chest*, *Critical Care Medicine*, and *Intensive Care Medicine*. Thanks to the AARC's steady increase in membership during the last two decades, its journal now reaches a larger number of hospitals and other health care facilities, and is received by more practitioners, than any other respiration-related scientific publication. This means that its potential for disseminating research results and other new information in areas important to clinicians is unmatched.

Where Has The Journal Fallen Short?

It has failed to get into ***Index Medicus***. This statement is misleading in that it implies that the responsibility for this failure rests with the Journal; it should be restated as "*Index Medicus* has failed to accept RESPIRATORY CARE into its listings." In the 14 years that I served on the Editorial Board the Journal applied to *Index Medicus* at every possible opportunity, and made every effort imaginable to satisfy the requirements of the National Library of Medicine and to convince the physicians on the *Index Medicus* Editorial Board that this "allied health" publication should be indexed. Every effort has failed.

The reasons for the denials have not been revealed, but their consequences can be. The Journal has clearly suffered in terms of manuscript submissions and physician attitudes about its contents; it is possible that patient care has also been adversely affected. For example, clinicians seeking assistance with patient management issues via MEDLINE cannot access relevant articles in RESPIRATORY CARE. Neither can investigators in the field. That the latter is the case is shown by the appearance in other journals of studies that duplicate previous studies published in RESPIRATORY CARE, and reviews that omit published work important to the topic under discussion. Despite the fact that the *Cumulative Index of Nursing and Allied Health Literature* (CINAHL), which indexes RESPIRATORY CARE, is now available on-line alongside MEDLINE, it is unlikely that all clinicians (or potential investigators) will search it when they should.

Academic promotion committees rely heavily on indexed publications when considering individual faculty members for promotion. Because of this, investigators in the field who are members of academic departments experience great pressure to submit their work to other journals if they can expect to achieve promotion. This results in substantial numbers of original research articles that should be published in RESPIRATORY CARE by virtue of their subject matter and intended audience being sent elsewhere.

The failure of *Index Medicus* to accept RESPIRATORY CARE for indexing surely cannot be justified on grounds of editorial standards and peer review. Anyone who has submitted research papers both to this journal and to its indexed sister pub-

lications can vouch for this. Especially in recent years, the process of reviewing and revising manuscripts is, if anything, more rigorous for RESPIRATORY CARE than for other journals.

Despite its absence from *Index Medicus*, the Journal's articles are cited by an ever-increasing number of authors in the field, both in research reports and in reviews, editorials, and book chapters. This fact attests to the acceptance of the work published in these pages by the field's leaders, and underscores the need to make it fully accessible to all workers in respiratory care and related fields through more appropriate indexing.

What Has Meant the Most to Me?

More than anything else, a publication such as RESPIRATORY CARE is a reflection of the people who produce it. And without doubt, it is the Journal's people that have made my interactions with it most meaningful—especially Phil Kittredge (who put RESPIRATORY CARE on the map, clinically and scientifically), Pat Brougher (who has expanded the scope and tightened the standards for the Journal's contents), and Ray Masferrer (whose vision and administrative skills have guided its course firmly and wisely from behind the scenes). During the years I have been associated with the Journal, it has been a genuine privilege to work with these outstanding individuals.

The Editorial Board also plays an important role. In my experience, members of the editorial boards of other journals serve primarily as manuscript reviewers and occasional editorialists—that is, to assure the quality of the journal's contents. In contrast, serving on the Editorial Board of RESPIRATORY CARE carries with it a heavy responsibility to help **create** the Journal's contents. Most of the innovations of the last 15 years—the Journal conferences, the institutional special issues, and the specialty features now found in each issue, for example—came out of Editorial Board meetings, and a substantial percentage of the contents of each year's volume is written or co-authored by members of this Board. To a considerable degree, the Journal's present structure, appearance, and direction reflect the efforts of the members of its Editorial Board.

RESPIRATORY CARE—who needs it? I do—and so does everyone else who cares about this field. The Journal has contributed in crucial ways to my own interest, persistence, and personal commitment to respiratory care. It has been nurtured and sustained by quality people, and its mission is important. Even in this era of professional uncertainty and economic constraints in health care, its future is sure to be one of continued achievement, unwavering quality standards, and increasing influence. I have high hopes for a continued long and fruitful relationship with RESPIRATORY CARE.

REFERENCES

1. Hudson LD. Monitoring of critically ill patients: conference summary. Respir Care 1985;30(7):628-636.
2. Pierson DJ. Respiratory care as a science. Respir Care 1988;33(1): 27-37.
3. Pierson DJ. The new horizons symposium: the first decade (foreword). Respir Care 1995;40(3):225-227.

Call for Abstracts

1996 RESPIRATORY CARE OPEN FORUM

Early Deadline: February 11, 1996
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Accepted abstracts will be printed in the
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Selected authors will present
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San Diego, California

Ureaplasma urealyticum Tracheal Colonization and Respiratory Disease in Newborns

Leandro Cordero MD, Kim Davis MT, Douglas Warner MD, and Leona W Ayers MD

BACKGROUND: Causality between *Ureaplasma urealyticum* (*U*) grown from tracheal cultures (TC) and bronchopulmonary dysplasia (BPD), pulmonary hypertension (PPHNB), and neonatal mortality has been proposed. We re-examined these relationships using a cohort design. MATERIALS & METHODS: We prospectively obtained TC from 377 mechanically ventilated infants delivered at University Hospital who were intubated an average of 23 days, and from whom we collected 783 TC (288 of them taken at birth and the rest weekly). Clinical and laboratory information was retrieved from medical records. RESULTS: Of the 377 infants, 54 (14%) had one or more positive TC for *U* during hospitalization. Of the 54 colonized infants, 45 (83%) weighed ≤ 1,250 g, 7 (13%) between 1,251 and 2,500 g, and 2 (4%) 2,500 g. One hundred and eighty four infants had birthweights ≤ 1,250 g. Forty-five (24%) of them were *U* positive and 139 (76%) were negative. Mean birthweight, gestational age, duration of mechanical ventilation, incidence and severity of BPD, and neonatal mortality were similar between the two groups. Forty infants diagnosed with PPHNB who were mechanically ventilated an average of 7 days provided 91 TC. Only 2 of these infants were *U* positive. Thirty-nine PPHNB patients survived and did not develop BPD. CONCLUSIONS: *U. urealyticum* tracheal colonization defined by serial TC is common among small, premature, mechanically ventilated infants. No association between this colonization and incidence or severity of BPD, neonatal mortality, and PPHNB was found. [Respir Care 1996;41(1):22-29]

Background

Since 1976, when *Ureaplasma urealyticum* (*U. urealyticum*) was isolated from 42 lungs of 290 stillborn infants,[1] to the present, our understanding of the role of these organisms in health and disease has increased significantly.[2] Of the many mycoplasmas that can be isolated from the lower genital tract of pregnant women only *U. urealyticum* and *Mycoplasma hominis (M. hominis)* have been implicated in disease of newborns.[3] Major contributions made by Cassell et al to our knowledge of perinatal mycoplasma infections are discussed in two excellent review articles.[3,4] Colonization of newborns is related to organism prevalence in the maternal genital tract, but not necessarily to the integrity of the amniotic membranes or to vaginal or cesarean delivery.[5]

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In 1991 Cassell et al[3] stated that although prospective studies have failed to show a definite role for *U. urealyticum* in neonatal respiratory disease, well-documented case reports have removed all doubts that this organism is capable of producing clinical and histologic pneumonia. The isolation of *U. urealyticum* from the lower respiratory tract of three infants with persistent pulmonary hypertension of the newborn (PPHNB) opened a new area of investigation.[6] Most of the scientific attention, however, has centered on the association of *U. urealyticum* colonization of the nasopharynx and/or the trachea of small premature infants and the development of bronchopulmonary dysplasia (BPD).[7-14] In a recent evaluation of four cohort studies, Wang et al[15] concluded that strong but not definite evidence shows that *U. urealyticum* causes BPD in premature infants. It should be noted that in three of these four studies ureaplasma samples were taken during the first days of life from the nasopharynx rather than from the trachea and that patients were characterized as colonized or noncolonized on the basis of only one positive or one negative culture.[7,9,10]

The purpose of the present cohort study was to correlate the clinical course of mechanically ventilated neonates with serially obtained TA cultures for *U. urealyticum*. A major objective was to determine whether associations existed between *U. urealyticum* tracheal colonization in small premature infants (birthweight ≤ 1,250 g) and neonatal mortality, PPHNB, and the incidence of BPD.

Materials & Methods

Patients Studied

Three hundred seventy-seven infants born at University Hospital between 1992 and 1994 who were admitted to the neonatal intensive care unit and who were intubated longer than 1 day comprised the study population. All infants required mechanical ventilation with a neonatal pressure-limited time-cycled ventilator.* Because of pulmonary interstitial emphysema or PPHNB, 9 of these infants were temporarily treated with high-frequency ventilation.*

All infants required umbilical-artery catheterization, and those who needed long-term intravenous therapy underwent central-venous catheterization. Exogenous surfactant was given at the manufacturer's recommended dosages. According to their clinical needs, infants with BPD were given dexamethasone sodium phosphate (0.5 mg kg^{-1} day^{-1} given in 2 doses q 12 h) intravenously for 3 days. Thereafter a 10% decrease in dosage every 3 days was initiated and continued for up to 4 weeks.[16,17]

Blood cultures, differential white-blood-cell counts (WBC-Dif), and chest radiographs were performed on all infants upon admission to the neonatal intensive care unit (NICU) and thereafter, as clinically indicated. WBC-Dif were done daily during the first week and bi-weekly thereafter. Subsequent blood cultures were taken whenever infection was suspected. All infants were treated prophylactically with ampicillin (100 mg · kg^{-1} · day^{-1} I.V., given in 2 doses q 12 h) and gentamicin (5 mg · kg^{-1} · day^{-1} I.V., given in 2 doses q 12 h). This regimen was continued for 3-5 days if the cultures were negative. Infants who developed sepsis late in their hospital course were initially treated with gentamicin and vancomycin. Once isolates were identified, the antibiotic therapy was changed according to the results of the antibiograms. BPD was defined by the clinical and radiologic signs as described by Northway et al[18] and by the need for supplemental oxygen at 28 days of life.[19] We characterized severity of BPD by the duration of mechanical ventilation in days and by the need for supplemental oxygen after discharge from the hospital. PPHNB was diagnosed by clinical, radiologic, and echocardiographic criteria.[20]

Microbial Colonization

To determine the presence or development of abnormal microbial colonization, weekly tracheal aspirates were obtained using aseptic technique. Normal saline (0.5 mL) was introduced into the endotracheal tube, suction applied, and the aspirated specimen used to saturate two sterile swabs. One swab for mycoplasma, ureaplasma, and chlamydia culture was immediately placed in 2SP transport media and placed on ice. The second swab was used for routine culture. Both swabs were immediately transported to the laboratory for processing or storage at 40° C overnight. If 2SP processing was delayed for longer than 24 hours, the transport tube was frozen at −70° C. The 2SP media was inoculated onto an A7B basal medium plate and into U9B broth for isolation of *U. urealyticum* and genital mycoplasmas and onto a cycloheximide-treated McCoy cell monolayer for recovery of *Chlamydia trachomatis (C. trachomatis)*. The inoculated agar plates were incubated in the presence of 5% CO_2 at 37° C for 48 hours, the broth culture in air at 37° C for 48 hours, and the monolayer in the presence of 5% CO_2 at 37° C for 48 hours. *Ureaplasma* and *Mycoplasma* spp. were identified by colony morphology and urease activity and *Chlamydia* sp. by a monoclonal antibody.[21]

The decision to treat one infant colonized with *U. urealyticum* was made empirically by an attending neonatologist. Erythromycin lactobionate 30 mg kg^{-1} day^{-1} was given intravenously in 4 divided dosages every 6 hours for 7 days, without improvement.

Data Collection & Statistical Analysis

Our NICU is managed under a prospective plan of patient care, which did not change over the study period. Results of

*Equipment and supplies are listed in the Product Sources Section at the end of the text.

the cultures were collected prospectively, and patient demographic and clinical information was obtained from hospital records. Subgroups were formed for specific analysis. The results are reported as mean (SD). Unpaired *t* tests were used to analyze the significance of the difference of group means for birthweight, gestational age, duration of intubation, and length of hospital stay. A χ^2 test on proportions was used for tracheal colonization, incidence of BPD, neonatal mortality, frequency of home oxygen supplementation, premature rupture of membranes (PROM), and mode of delivery.

A patient was considered colonized if at any time during hospitalization at least one tracheal culture yielded *U. urealyticum* and noncolonized if TC were always negative or if two consecutive TC taken within a week of each other were negative. Each infant whose birthweight was ≤ 1,250 g and who survived longer than 28 days was classified as negative (noncolonized n = 121) or positive (colonized n = 38). Forward stepwise logistic regression [22] was employed to assess the influence of race, birthweight, gestational age, gender, duration of ventilation, length of hospital stay, surfactant administration, dexamethasone treatment, and *U. urealyticum* colonization of the trachea on BPD.

Results

Demographics

Three hundred and sixteen (83%) of the 377 mechanically ventilated neonates were born before 37 weeks of gestation. The birthweight for the entire study population ranged from 430 to 4,770 g (Table 1). One hundred and eighty-four of these infants weighed ≤ 1,250 g. Sixty-one newborns who were born at more than 37 weeks of gestation weighed > 2,500 g. Three hundred and seventeen infants (84%) were singleton, 276 (73%) patients were Caucasian, 96 patients (25%) were Black, and 5 (1%) were Oriental. Of all neonates, 55% were male. Of the 377 infants, 214 (56%) were delivered by cesarean section.

Respiratory failure of nonpulmonary origin was diagnosed in 56 cases (15%), hyaline membrane disease in 271 (72%), PPHNB in 40 (11%), and pneumonia in 14 (5%). Exogenous surfactant was administered to 83% of the 377 infants. Sixty newborns who did not receive surfactant were older than 33 weeks of gestation and had birthweights ≥ 2,000 g. One hundred and twenty-five of 377 infants (36%) received treatment with dexamethasone not earlier than the second week of life.

U. urealyticum Tracheal Colonization

M. hominis was isolated in 3, and *C. trachomatis* in 8 of the 783 TC. None of these 11 infants had any clinical or radiologic evidence of pneumonia, and none of these organisms grew in subsequent TC. Concurrent growth of *U. urealyticum*, *M. hominis*, and *C. trachomatis* occurred in one and two TC respectively. During hospitalization, 54 of 377 newborns (14%) had one or more positive tracheal cultures for *U. urealyticum* (Fig 1). Only in 19 of the 54 (35%) colonized infants were all TC positive. The remaining 35 colonized neonates (65% of the entire group) had one or more negative TC sometime during the hospitalization. The clinical courses of the 19 patients with all positive cultures and those of the 35 with intermittent positives were similar.

TC were taken during the first hour of life in 288 newborns. Of these, 39 (14%) were *U. urealyticum* positive and 249 (86%) were negative. Of the infants with initially negative TC, 10 became positive at 1 week, 4 at 2 weeks, and 1 at 3 weeks of life. In 15 instances (6%) the original TC culture did not characterize the infants as colonized, and in 6 of these cases 2 negative cultures were obtained before the first positive TC was encountered.

Tracheal colonization decreased with increasing gestational age and birthweight (Table 1). Thirty percent of 47 infants who weighed under 750 g, 26% of 84 who weighed between 751 and 1,000 g and 17% of 53 newborns who weighed between 1,001 and 1,250 g were *U. urealyticum* positive.

Table 1. Population Studied To Determine Factors Related to *U. urealyticum* Tracheal Colonization

Birthweight (g)	Number of Patients	Gestational Age (weeks)	Duration of Intubation (days)	Length of Stay (days)	Patients Dead number (%)	Ureaplasma Positive number (%)
≤ 750	47	26 (1)*	39 (21)*	67 (37)*	11 (23)	14 (30)
751-1,000	84	27 (2)	34 (19)	67 (24)	12 (14)	22 (26)
1,001-1,250	53	29 (2)	19 (19)	50 (16)	4 (4)	9 (17)
1,251-1,500	42	31 (2)	10 (8)	38 (13)	1 (2)	3 (7)
1,501-1,750	23	32 (20)	4 (8)	25 (10)	0	0 (0)
1,751-2,000	20	33 (2)	5 (6)	21 (8)	0	1 (5)
2,001-2,250	30	34 (1)	6 (7)	18 (11)	0	2 (6)
2,251-2,500	17	35 (1)	5 (6)	13 (6)	0	1 (6)
> 2,500	61	38 (2)	4 (2)	10 (5)	1 (3)	2 (3)
Total	377	31 (4)	15 (18)	41 (30)	29 (7)	54 (14)

*Values are mean (SD).

Birthweight (g)	Weeks on Mechanical Ventilation: Birth	1	2	3	4	5	6	7	8	9
430	•	•	+	+	+	•	•	NST		
607	NST	NST	+	•	•	•	•			
620	•	•	+	•	•	•	NST			
660	NST	+	+							
670	•	+	+	+	+	+				
680	+	•	+	+	+	+	+			
680	NST	+	+	•	•	•	•	•		
680	+	•	+	•	•	+	+	NST		
680	+	NST	+	NST	NST					
700	+	+	NST	+	NST					
700	•	+	+							
700	NST	NST	+	•	•	+				
710	+	NST								
720	•	+	•	•	•	•	+	+	NST	NST
759	•	+	+	•	+	NST				
760	+	+	+	•	•	•	•	•		
760	NST	+	+	NST						
780	•	+	+	NST						
789	NST	+	+	NST						
830	+	+	+	NST						
840	•	•	+	+						
850	+	+	NST							
850	+	+	NST							
850	+	+	+	NST	+					
850	•	+	NST	•						
860	NST	+	+	+	+	+	+	+		
860	•	+	+	NST						
908	NST	NST	+	+	+	NST	•	NST	•	
910	•	+	+	NST						
920	•	•	+	•	+					
930	NST	NST	+	+	NST					
935	•	•	+	NST	+					
940	NST	+	+	NST						
970	NST	+	•	+						
980	+	NST								
1000	NST	•	+							
1040	+	•	•	•						
1045	+	+	NST							
1060	+	NST								
1070	+	NST								
1100	+	NST	•	•						
1140	NST	•	+	+						
1190	+	•	+	+						
1250	NST	+	+							
1250	+	NST	NST	•	•					
1380	NST	•	+							
1396	+	NST	•							
1500	•	+	+							
1800	+	+								
2100	NST	•	+							
2160	+	+	+	•						
2320	NST	•	•	+						
3185	+									
3880	+	NST								

Fig. 1. Tracheal cultures from 54 *U. urealyticum* colonized infants. • = negative, + = positive, NST = no sample taken.

Nineteen of 20 sets of twins yielded negative TC. Both members of the remaining set were TC positive for *U. urealyticum*. Four of five quintuplets delivered by cesarean section who were tested had negative TC. Only one member of another 16 sets of twins was available for airway culture. Three of these 16 infants were TC positive. Thus, for the entire group of 60 newborns who were products of multiple pregnancies, only 5 (8%) were *U. urealyticum* colonized.

U. urealyticum airway colonization was more common among infants born vaginally than among those delivered by cesarean section (Table 2). Prolonged rupture of membranes (PROM) was also associated with an increase in airway colonization. Sixty percent of TC-positive and 40% of TC-negative infants were delivered vaginally (χ^2 8.27, $p < 0.01$). Ten TC positive infants were born by cesarean section in the presence of intact membranes.

Table 2. *U. urealyticum* Tracheal Colonization, Duration of Rupture of Amniotic Membranes (ROM), and Mode of Delivery.

	Vaginal Infants Colonized n	n (%)	Cesarean Infants Colonized n	n (%)	p Value*
ROM ≤ 24 h	97	16 (16)	164	13 (8)	0.05
ROM > 24 h	66	18 (27)	50	7 (14)	0.30
Total	163	34 (21)	214	20 (9)	0.002

*$\chi^2_{n=1,0.95}$

Neonatal Mortality

Of the 377 patients, 29 (7%) died during the neonatal period or before the sixth month of life. Mortality figures for 184 infants with birthweights ≤ 1,250 g are presented in Table 3. Six of 45 (13%) *U. urealyticum* positive infants, and 21 of the 139 (15%) negative infants died. This difference in mortality is not statistically significant ($p = 0.09$). Two of the 193 patients whose birthweight was > 1,250 g also died, although neither was *U. urealyticum* positive.

Persistent Pulmonary Hypertension

Forty neonates who developed respiratory failure were diagnosed with PPHNB. Birthweight ranged from 2,480 to 3,880 g ($\bar{x}$ = 2,996), hospital stay from 6 to 65 days ($\bar{x}$ = 17) and duration of mechanical ventilation from 3 to 22 days ($\bar{x}$ = 7). Clinical correlates were meconium aspiration syndrome (5 patients), PROM and oligohydramnios (7 patients), acute fetal distress (5 patients), chronic abruptio placenta (4 patients), placenta previa (4 patients), Group-B streptococcus sepsis (2 patients), and congenital heart disease (2 patients). In the remaining patients, the cause of PPHNB remained unclear. All infants required conventional mechanical ventilation supplemented, in 6 cases, with high frequency oscillation and, in 2, with extracorporeal membrane oxygenation. Of the entire group, only one infant diagnosed with congenital heart disease died. Thirty-eight of the 40 TC taken during the first hour following birth were negative. Thirty-seven of these cultures were repeated within 1 week and remained negative. Thirteen additional cultures taken 1 week later were also negative. Of the 2 patients with TC positive at birth, one was extubated at 3 and the other at 10 days (in this particular patient a second culture was also positive).

Table 3. *U. urealyticum* Tracheal Colonization and Neonatal Mortality.

Birthweight (g)	Positive Patients n	Positive Patients Dead (%)	Negative Patients n	Negative Patients Dead (%)	Total Patients	p Value*
≤ 750	14	3 (21)	33	8 (24)	47	0.9
751-1,000	22	3 (14)	62	9 (15)	84	0.8
1,001-1,250	9	0 (0)	44	4 (9)	53	0.6
Total	45	6 (13)	139	21 (15)	184	0.9

*$\chi^2_{n-1,0.95}$

Tracheal Colonization & BPD

One hundred and fifty-nine infants with birthweights ≤ 1,250 g, who survived 28 days or who died after the diagnosis of BPD was established were included in this analysis (Table 4). The incidence of BPD ranged from 94% for infants who weighed 1,000 g to 59% for those with birthweights between 1,001 and 1,250 g. For the total group, 92% of positive and 80% of TA negative newborns developed BPD. This difference was not statistically significant ($p = 0.14$). Stepwise logistic regression analysis established that clinical variables associated with BPD were duration of ventilation ($p = 0.0001$), gestational age ($p = 0.0001$), and birthweight ($p = 0.0001$). After controlling for these factors, none of the other variables studied, including *U. urealyticum*, were related to BPD. The association between *U. urealyticum* colonization and BPD was not statistically significant ($p = 0.13$). Two additional logistic regression analyses, one which did not include duration of ventilation and one that included higher order interactions with *U. urealyticum*, produced the same results.

For infants with birthweight ≤ 1,250 g, the average duration of ventilation was 34 and 31 days for *U. urealyticum* positive and negative infants respectively. Forty-seven percent of TA positive and 51% of TA negative infants were discharged home on supplemental oxygen. None of these differences was statistically significant ($p = 0.8$).

Discussion

Of the many mycoplasmas that can be isolated from the lower genital tract of pregnant women only *U. urealyticum* and *M. hominis* have been implicated in diseases of the newborn.[3] *M. hominis* has been recovered from infants with respiratory distress.[23] The low recovery in our study population is not unexpected because in similar groups of small premature infants mycoplasma prevalence in TC ranged from 2 to 7%.[8,14]

Blood, cerebrospinal fluid, nasopharynx (NP), gastric aspirate, pleural fluid, lung tissue, and tracheal secretions are all suitable for *U. urealyticum* culture.[2] Samples taken from sites other than the trachea are less reliable in evaluating lung infections because they may not represent the condition of the lung itself. Even airway cultures should be interpreted cautiously because tracheal specimens were negative in 2 patients whose pleura and lung biopsies yielded *U. urealyticum*.[24] The administration of exogenous surfactant to our patients did not interfere with the recovery of *U. urealyticum* from the trachea.[10]

In 1988 Cassell et al,[8] who studied nasopharyngeal and TC samples in 200 prematures, concluded that recovery of ureaplasma from endotracheal aspirates did not correlate with its presence in the nasopharynx. Payne et al[10] studied 93 premature infants from whom nasopharyngeal and endotracheal cultures were taken at about 2 and 14 days of life. This group reported that most TC remained positive while almost all NP became negative. Further discrepancies have been highlighted by Johnson et al,[11] who found that in 19 cases of ureaplasma colonization 9 infants were NP and TC positive, 5 were only TC positive, and 4 were only NP positive. In spite of the above, investigators have reported colonization rates to be associated with BPD, regardless of the origin of the sample.[7-10,12,15,25]

Characterizing newborn populations as colonized or noncolonized on the basis of a single NP or TA culture is problematic. We have demonstrated that 7% of initially negative TC would have been misleading unless repeated within 2 weeks.

Table 4. *U. urealyticum* Tracheal Colonization and the Incidence of Bronchopulmonary Dysplasia (BPD)

Birthweight (g)	Positive Patients n	Positive Patients BPD (%)	Negative Patients n	Negative Patients BPD (%)	Total Patients	p Value*
≤ 750	12	12 (100)	25	24 (96)	37	0.7
751-1,000	19	19 (100)	51	46 (90)	70	0.3
1,001-1,250	7	4 (57)	45	27 (60)	52	0.7
Total	38	35 (92)	121	97 (80)	159	0.14

*$\chi^2_{n-1,0.95}$

To our knowledge only Heggie et al.[14] like ourselves, determined noncolonization after 2 negative TC. Because nosocomial infections have not been demonstrated,[3,4] we may assume that factors such as antibiotic exposure, level of colonization, adequacy of sampling, or cultivation techniques[26] explain inconsistently positive cultures. Samples obtained weekly have allowed us to study 19 infants whose TC were always positive and 35 infants with intermittent TC positivity. Although we did not observe clinical differences among these two groups of patients, further investigation is highly warranted.

Tracheal colonization developed in infants born vaginally as well as in those delivered by cesarean section regardless of the condition of the amniotic membranes.[8] Our observation of 10 TC-positive infants born by cesarean delivery with intact amniotic membranes followed that pattern. Although PROM has not been correlated with *U. urealyticum* colonization,[5,10,11,27] its more common occurrence among vaginally delivered infants in the present study is of interest considering that infection of the chorioamnion is independent of the onset and duration of labor and the duration of rupture of the amniotic membranes.[28]

Tracheal colonization with *U. urealyticum* among infants with birthweight ≤ 1,000 g had been associated with a mortality of 34% among 56 noncolonized and 70% for 20 colonized infants.[8] This observation differed with that reported here (18% for 95 noncolonized and 17% for 36 colonized individuals). It is unclear whether the extremely high mortality for the patients in the study by Cassell and colleagues[8] was due to an unusually large number of very small infants or to epidemiological, demographic, or medical differences. Recently, Heggie et al[14] who studied *U. urealyticum* tracheal colonization in 54 infants with birthweights ≤ 1,500 g reported mortalities of 10 and 8% for 30 colonized and 24 noncolonized infants respectively.

Persistent pulmonary hypertension of the newborn is a complex disorder of multiple etiology characterized by increased vascular resistance that leads to shunting through the foramen ovale, ductus arteriosus, or both.[18] Experimental work has linked Group-B streptococcus as well as gram-negative bacteria to pulmonary hypertension.[29-32] In 1989, Waites et al[6] reported 3 cases of PPHNB from whom *U. urealyticum* was recovered from the trachea, and concluded that this organism, like streptococci, should be considered a cause or, at least, a contributing etiologic factor. If this association were valid, it would be reasonable to expect significant *U. urealyticum* colonization among PPHNB patients. Instead only 2 out of 40 patients proved to be tracheally colonized, and, even more important, all noncolonized patients remained negative one week later. This was not totally unexpected because PPHNB seldom affects infants of gestational age less than 33 weeks—precisely the group in whom *U. urealyticum* airway colonization is more common.

Bronchopulmonary dysplasia is a chronic lung condition that affects mainly small, premature infants who have been treated with positive pressure ventilation and oxygen.[33] Advances in conventional and high-frequency ventilation and the availability of surfactant have increased survival of critically ill, low-birthweight neonates with a resulting increase in the number of infants with BPD.[34] Long-term complications of BPD, such as respiratory insufficiency, pulmonary and systemic hypertension, growth retardation, and respiratory infections, still occur with predictable frequency. The increased incidence of sudden-infant-death syndrome reported among infants with BPD has generated widespread concern and controversy.[35-37]

The incidence of BPD varies according to the definition chosen and can be as high as 100% for infants with birthweight < 800 g or as low as 5% for those with birthweight > 1,500 g. Many factors such as demographics, place of birth, and level of medical and nursing care, also result in different rates of neonatal mortality and BPD.[38,39]

Lung injury by hyperoxia, barotrauma, and infection with the subsequent inflammatory and reparative response is the factor most strongly associated with the development of BPD. Direct tissue damage can be potentiated by the activation of inflammatory cells and chemicals.[33,40] Theoretical causal links between BPD and *U. urealyticum* have been made by showing that human neonatal pulmonary fibroblasts release significant levels of interleukins-5 and -8 in direct response to infections.[41]

U. urealyticum specifically enhances the production of tumor necrosis factor (alpha), a potent proinflammatory cytokine produced by macrophages, and is thought to play a critical role in the development of lung disease.[42] Because severe and persistent lung disease has been associated with infectious agents such as cytomegalovirus and chlamydia,[43,44] it is natural to question whether *U. urealyticum* participates in the acute injury phase of BPD. Mice infected with *U. urealyticum* and exposed to hyperoxia experience longer infections and higher mortality rate than the controls.[45] In an animal study, inoculation of premature baboon infants with *U. urealyticum* results in recognizable, albeit mild, pulmonary lesions.[46]

Causality between clinical BPD and *U. urealyticum* in the infant airway, however, remains unproven. This organism has been implicated clinically by temporal association with BPD infants using cohorts with colonized and noncolonized airways defined by single nasopharyngeal samples.[7,9,12] With some exceptions,[8-10] investigations including ours, which focused mainly on serial tracheal cultures, found no such association.[11,13,14] Payne et al[10] defined severity of BPD using a radiological score and concluded that there was no evidence that colonized infants developed more severe BPD than those not colonized. Using different criteria for assessing severity of BPD, we arrived at a similar conclusion.

Because tracheal cultures may be insufficient to define the study populations of cohorts, infants with tracheal ureaplasma detected by a single culture immediately after delivery may represent an entirely different risk group than infants who

demonstrate tracheal ureaplasma in the weeks following birth. Early detection of tracheal ureaplasma with techniques such as polymerase chain reaction may define more quickly which infants are colonized, but may not necessarily better predict those at risk for infection-enhanced lung disease.[47-49] Attention may need to be turned to the use of blood and/or spinal fluid for *U. urealyticum* culture or to serologic studies[50,51] to differentiate between infected and colonized infants.

Randomized placebo-controlled clinical trials of erythromycin therapy have been proposed as a means to establish the relationship between *U. urealyticum* and newborn lung disease.[15,52,53] Such studies appear premature in view of the conflicting reports of erythromycin's clinical efficacy in small unblinded trials.[14,54-58] Erythromycin is reported to be relatively safe for infants but does not penetrate the central nervous system and, although active against most strains, has been irregular in the complete eradication of the organism from the respiratory tract.[52,53] Despite the narrow spectrum of antibiotics active against *U. urealyticum*, prenatal treatment of the mother with macrolide or tetracycline antibiotics or treatment of the low-birthweight infant with gentamicin, a partially active antibiotic, could affect initial recovery of ureaplasma from infected and colonized infants.[59] Except in the rare cases of documented clinical infections, the parameters for selection of low-birthweight infants who are likely to profit from antibiotic therapy remain undefined.

Conclusions

U. urealyticum tracheal colonization defined by serial tracheal cultures is common among mechanically ventilated premature infants with birthweight ≤ 1,250 g. No significant statistical association between airway colonization and neonatal mortality, PPHNB, and incidence and severity of BPD was found. Tracheal cultures taken at birth and weekly thereafter should be obtained before characterizing newborns as colonized or not colonized. Otherwise, misleading epidemiologic conclusions could be drawn.

PRODUCT SOURCES

Conventional Mechanical Ventilator:
Sechrist IV B Sechrist Industries Inc, Anaheim CA

High-Frequency Flow Interrupter:
Infant Star Neonatal High Frequency Ventilator, Infrasonics Inc, San Diego CA

BabyLog 8000:
Drager Inc, Critical Care Systems, Chantilly VA

3100 A Oscillator:
SensorMedics Critical Care, Yorba Linda CA

Pulmonary Surfactant:
Exosurf, Burroughs Wellcome Co, Research Triangle Park NC
Survanta, Ross Laboratories, Columbus OH
Infasurf, Forest Laboratories, St. Louis MO

Central Venous Catheter:
Broviac Single-Lumen Vascular Access Catheter, Bard Access Systems, Salt Lake City UT

Statistical Software:
BMDP Statistical Software Inc, Los Angeles CA

REFERENCES

1. Tafari N, Ross S, Naeye RL, Judge DM, Marboe C. Mycoplasma T strains and perinatal death. Lancet 1976;1(7951):108-109.
2. Waites KB, Crouse DT, Cassell GH. Systemic neonatal infections due to *Ureaplasma urealyticum*. Clin Infect Dis 1993;17(Suppl 1): 131S-135S
3. Cassell GH, Waites KB, Crouse DT. Perinatal mycoplasmal infections. Clin Perinatol 1991;18(2):241-262.
4. Cassell GH, Waites KB, Watson HL, Crouse DT, Harasawa R. *Ureaplasma urealyticum* intrauterine infection: role in prematurity and disease in newborns. Clin Microbiol Rev 1993;6(1):69-87.
5. Sanchez PJ. Perinatal transmission of *Ureaplasma urealyticum*: current concepts based on review of the literature. Clin Infect Dis 1993;17(Suppl 1):107S-111S.
6. Waites KB, Crouse DT, Philips JB 3d, Canupp KC, Cassell GH. Ureaplasmal pneumonia and sepsis associated with persistent pulmonary hypertension of the newborn. Pediatrics 1989;83(1):79-85.
7. Wang EE, Frayha H, Watts J, Hammerberg O, Chernesky MA, Mahony JB, Cassell GH. Role of *Ureaplasma urealyticum* and other pathogens in the development of chronic lung disease of prematurity. Pediatr Infect Dis J 1988;7(8):547-551.
8. Cassell GH, Waites KB, Crouse DT, Rudd PT, Canupp KC, Stagno S, Cutter GR. Association of *Ureaplasma urealyticum* infection of the lower respiratory tract with chronic lung disease and death in very-low-birth-weight infants. Lancet 1988;2(8605):240-245.
9. Sanchez PJ, Regan JA. *Ureaplasma urealyticum* colonization and chronic lung disease in low birth weight infants. Pediatr Infect Dis J 1988;7(8):542-546.
10. Payne NR, Steinberg SS, Ackerman P, Chrenka BA, Sane SM, Anderson KT, Fangman JJ. New prospective studies of the association of *Ureaplasma urealyticum* colonization and chronic lung disease. Clin Infect Dis 1993;17(Suppl 1):117S-121S.
11. Jonsson B, Karell A-C, Ringertz S, Rylander M, Faxelius G. Neonatal *Ureaplasma urealyticum* colonization and chronic lung disease. Acta Paediatr 1994;11(1):33-36.
12. Horowitz S, Landau D, Shinwell ES, Zmora E, Dagan R. Respiratory tract colonization with *Ureaplasma urealyticum* and bronchopulmonary dysplasia in neonates in southern Israel. Pediatr Infect Dis J 1992;11(10):847-851.
13. Cordero L, Davis K, Warner D, Ayers LW. *Ureaplasma Urealyticum* Tracheal Colonization of Mechanically Ventilated Newborn (abstract). Pediatr Res 1994;35:267A.
14. Heggie AD, Jacobs MR, Butler VT, Baley JE, Boxerbaum B. Frequency and significance of isolation of *Ureaplasma urealyticum* and *Mycoplasma hominis* from cerebrospinal fluid and tracheal aspirate specimens from low birth weight infants. J Pediatr 1994;124(6):956-961.
15. Wang EE, Cassell GH, Sanchez PJ, Regan JA, Payne NR, Liu PP. *Ureaplasma urealyticum* and chronic lung disease of prematurity: critical appraisal of the literature on causation. Clin Infect Dis 1993; 17(Suppl 1):112S-116S.
16. Avery GB, Fletcher AB, Kaplan M, Brudno DS. Controlled trial of dexamethasone in respirator-dependent infants with bronchopulmonary dysplasia. Pediatrics 1985;75(1):106-111.
17. Harkavy KL, Scanlon JW, Chowdhry PK, Grylack LJ. Dexamethasone therapy for chronic lung disease in ventilator and oxygen-dependent infants: a controlled trial. J Pediatr 1989;115(6):979-983.

18. Northway WH Jr, Rosan CR, Porter DY. Pulmonary disease following respiratory therapy of hyaline-membrane disease. N Engl J Med 1967; 276(7):357-368.
19. Bancalari E, Abdenour GE, Feller R, Gannon J. Bronchopulmonary dysplasia: clinical prevention. J Pediatr 1979;95(5, Part 2):819-823.
20. Gersony WM. Neonatal pulmonary hypertension: pathophysiology, classification, and etiology. Clin Perinatol 1984;11(3):517-524.
21. Clyde, WA, Kenny GE, Schacter JA. Laboratory diagnosis of chlamydial and mycoplasmal infections. Cunitech 19. Washington DC: American Society for Microbiology, 1984.
22. Afifi AA, Clark V. Computer-aided multivariate analysis. Belmont CA: Lifetime Learning Publications, 1990.
23. Brunell PA, Dische RM, Walker MB. Mycoplasma, ammionitis, and respiratory distress syndrome. JAMA 1969;207:2097-2099.
24. Walsh WF, Stanley S, Lally K, Stribley RE, Treece DP, McCleskey F, Null DM. *Ureaplasma urealyticum* demonstrated by open lung biopsy in newborns with chronic lung disease. Pediatr Infect Dis J 1991; 10(11):823-827.
25. Wang EEL, Ohlsson A, Kellner JD. Association of *Ureaplasma urealyticum* colonization with chronic lung disease of prematurity: Results of a meta-analysis. J Pediatr 1995;127:640-644.
26. Waites KB, Duffy LB, Searcey KB, Cassell GH. Importance of broth-to-agar inoculation and serial dilution for isolating *Ureaplasma urealyticum* from endotracheal aspirates (abstract). Program and Abstracts from the Annual Meeting, American Society of Microbiology, Washington DC 1995;300.
27. Syrogiannopoulos GA, Kapatais-Zoumbos K, Decavalas GO, Markantes CG, Katsarou VA, Beratis NG. *Ureaplasma urealyticum* colonization of full term infants: prenatal acquisition and persistence during early infancy. Pediatr Infect Dis J 1990;9(4):236-240.
28. Eschenbach DA. *Ureaplasma urealyticum* and premature birth. Clin Infect Dis 1993;17(Suppl 1):100S-106S.
29. Rojas J, Stahlman M. The effect of group B streptococcus and other organisms on the pulmonary vasculature. Clin Perinatol 1984;11(3): 591-599.
30. Gibson RL, Truog WE, Redding GJ. Nonspecific thromboxane mediated pulmonary hypertension in neonatal Gram-positive sepsis (abstract). Am Rev Respir Dis 1987;135:124.
31. Shankaran S, Farooki ZQ, Desai R. β-Hemolytic streptococcal infection appearing as persistent fetal circulation. Am J Dis Child 1982;136(8):725-727.
32. Kinsella JP, Abman SH. Recent developments in the pathophysiology and treatment of persistent pulmonary hypertension of the newborn. J Pediatr 1995;126(6):853-864.
33. Abman SH, Groothius JR. Pathophysiology and treatment of bronchopulmonary dysplasia. Pediatr Clin North Am 1994;41(2):277-313.
34. Parker RA, Lindstrom DP, Cotton RB. Improved survival accounts for most, but not all of the increase in bronchopulmonary dysplasia. Pediatrics 1992;90(5):663-668.
35. Werthammer J, Brown ER, Neff RK, Taeusch, HW Jr. Sudden infant death syndrome in infants with bronchopulmonary dysplasia. Pediatrics 1982;69(3):301-304.
36. Meny RG, Blackmon L, Fleischmann D, Gutberlet R, Naumburg E. Sudden infant death and home monitors. Am J Dis Child 1988;142 (10):1037-1040.
37. Gray PH, Rogers Y. Are infants with bronchopulmonary dysplasia at risk for sudden infant death syndrome? Pediatrics 1994;93(5): 774-777.
38. Shennan AT, Dunn MS, Ohlsson A, Lennox K, Hoskins EM. Abnormal pulmonary outcomes in premature infants: prediction from oxygen requirement in the neonatal period. Pediatrics 1988;82(4): 527-532.
39. Avery ME, Tooley WH, Keller JG, Hurd SS, Bryan MH, Cotton RB, et al. Is chronic lung disease in low birth weight infants preventable? A survey of eight centers. Pediatrics 1987;79(1):26-30.
40. Stenmark KR, Eyzagirre M, Westcott JY, Henson PM, Murphy RC. Potential role of eicosanoids and platelet activating factor in the pathophysiology of BPD. Am Rev Respir Dis 1987;136(3):770-772.
41. Stancombe BB, Walsh WF, Derdak S, Dixon P, Hensley D. Induction of human neonatal pulmonary fibroblast cytokines by hyperoxia and *Ureaplasma urealyticum*. Clin Infect Dis 1993;17(Suppl 1): 154S-157S.
42. Crouse DT, English K. Mycoplasma induction of TNF alpha from RAW 264.7 cells (abstract). Pediatr Res 1995;37:329A
43. Rudd PT, Carrington D. A prospective study of chlamydial mycoplasmal, and viral infections in a neonatal intensive care unit. Arch Dis Child 1984;59(2):120-125.
44. Stagno S, Brasfield DM, Brown MB, Cassell GH, Pifer LL, Whitley RJ, Tiller RE. Infant pneumonitis associated with cytomegalovirus, *Chlamydia*, *Pneumocystis*, and *Ureaplasma*: a prospective study. Pediatrics 1981;68(3):322-327.
45. Crouse DT, Cassell GH, Waites KB, Foster JM, Cassady G. Hyperoxia potentiates *Ureaplasma urealyticum* pneumonia in newborn mice. Infect Immun 1990;58(11):3487-3493.
46. Walsh WF, Buttler J, Coalson J, Hensley D, Cassell GH, deLemos RA. A primate model of *Ureaplasma urealyticum* infection in the premature infant with hyaline membrane disease. Clin Infect Dis 1993; 17(Suppl 1):158S-162S.
47. Blanchard A, Hentschel J, Duffy L, Baldus K, Cassell GH. Detection of *Ureaplasma urealyticum* by polymerase chain reaction in the urogenital tract of adults, in amniotic fluid and in the respiratory tract of newborns. Clin Infect Dis 1993;17(Suppl 1):148S-153S.
48. Scheurlen W, Frauendienst G, Schrod L, von Stockhausen HB. Polymerase chain reaction-amplification of urease genes: rapid screening for *ureaplasma urealyticum* infection in endotracheal aspirates of ventilated newborns. Eur J Pediatr 1992;151(10):740-742.
49. Quinn Pa, Li CH, Th'ng C, Dunn M, Butany J. Serological response to *Ureaplasma urealyticum* in the neonate. Clin Infect Dis 1993;17 (Suppl 1):136S-143S.
50. Lau K, Zheng X, Cassell GH, Watson HL. Identification of epitopes of a major antigen of *Ureaplasma urealyticum* (abstract). Pediatr Res 1995;37:294.
51. Toce SS, Farrell PM, Leavitt LA, Samuels DP, Edwards DK. Clinical and roentgenographic scoring systems for assessing bronchopulmonary dysplasia. Am J Dis Child 1984;138(6):581-585.
52. Waites KB, Crouse DT, Cassell GH. Therapeutic considerations for *Ureaplasma urealyticum* infections in neonates. Clin Infect Dis 1993; 17(Suppl 1):208S-214S.
53. Waites KB, Sims PJ, Crouse DT, Geerts MH, Shoup RE, Hamrick WB, et al. Serum concentrations of erythromycin after intravenous infusion in preterm neonates treated for *Ureaplasma urealyticum* infection. Pediatr Infect Dis J 1994;13(4):287-293.
54. Groenendaal F, Angulo AF. *Ureaplasma urealyticum* in tracheal aspirate of ventilated premature infants: report of 6 cases. Tijdschr Kindergeneeskd 1993;61(1):20-24.
55. Abele-Horn M, Hentschel J. *Ureaplasma urealyticum* in newborn and premature infants. Its association with bronchopulmonary dysplasia. Dtsch Med Wochenschr 1992;117(11):408-414.
56. Baier J, Fajardo C. Early screening and treatment of *U. urealyticum* in the VLBW infant does not prevent chronic lung disease (abstract). Pediatr Res 1993;35:273A.
57. Satow K, Groneck P, Schutt-Gerowitt H, Roth B, Benz-Bohm G. *Ureaplasma urealyticum*—a new problem pathogen in neonatology. Monatsschr Kinderheikd 1991;139(6):344-348.
58. Gjuric G, Prislin-Muskic M, Zurga B, Nikolic E, Cordasic R, Stanojevic M. *Ureaplasma urealyticum* infection in newborns: three case reports. Eur J Pediatr 1993;152(7):599-600.
59. Waites KB, Crouse DT, Cassell GH. Antibiotic susceptibilities and therapeutic options for Ureaplasma urealyticum infections in neonates. Pediatr Infect Dis J 1992;11(1):23-29.

Use of Pulse Oximetry To Determine Oxygen Prescription for Hypoxemic Patients with COPD

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BACKGROUND: A retrospective analysis was conducted to determine whether oxyhemoglobin saturation measured by pulse-oximetry (S_{pO_2}) during a 5-minute corridor walk ($WALK^{5:00}$) accurately predicts oxygen flow required by hypoxemic patients with chronic obstructive pulmonary disease (COPD) using the standard nasal cannula (SNC) and reservoir nasal cannula (RNC) to maintain average $S_{pO_2} \geq 91\%$ during simulated activities of daily living (ADLs). METHODS: Oxygen dose was titrated in 15 men who were hypoxemic (average $S_{pO_2} \leq 88\%$) at rest or with mild exercise, mean (SD) age 69 (9) years, with 40 (17) pack-year smoking history, $FEV_1/FVC\%$ 41 (10)%, P_{aO_2} 57 (4) torr, and exertional dyspnea. Each subject used both devices, on separate occasions, in a randomized order, during incremental cycle ergometry and a 4-station circuit simulating ADLs. RESULTS: Average S_{pO_2} during the last 30 seconds of $WALK^{5:00}$ ($WALK^{:30}$) was significantly greater than during the lift-push-carry, undress and dress, and ascending and descending stairs. No significant difference between average S_{pO_2} for $WALK^{:30}$ [SNC = 89 (3)%, RNC = 90 (3)%] and bicycle ergometry [SNC = 91 (3)%, RNC = 92 (3)%] was found. CONCLUSION: SNC and RNC oxygen flow prescriptions intended to maintain $S_{pO_2} \geq 91\%$ during physical exertion in hypoxemic COPD patients may be determined during a 5-minute corridor walk using only the average of S_{pO_2}s taken during the last 30 seconds of exercise. [Respir Care 1996;41(1):30-36]

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Introduction

A number of studies have justified the use of oxygen therapy during exercise in patients with hypoxemic chronic obstructive pulmonary disease (COPD).[1-11] In this population, oxyhemoglobin saturation frequently decreases during exercise.[1-14] Some COPD patients who have mild or no hypoxemia at rest may need oxygen therapy during exercise, and many hypoxemic patients require a higher oxygen flow during exercise. The need for a higher oxygen flow may be secondary to the presence of worsening ventilation-perfusion relationships and/or diffusion limitations or to the hyperpnea of exercise resulting in greater dilution of oxygen with room air.[12] Oxygen administration during exercise has been shown to improve exercise tolerance at submaximal workloads[2,3,6,7] and to reduce pulmonary artery pressure and dyspnea.[9-11]

Most studies evaluating exercise-induced hypoxemia have employed a bicycle, a treadmill-walk, or a 12-minute corridor-walk test.[13] Bicycle testing seriously underestimates exercise-induced oxyhemoglobin desaturation compared to the

treadmill-walk test at a similar level of work.[13,14] In COPD patients, step-test exercise has been found to produce even greater reductions in arterial oxygen tension than does steady-state treadmill walking.[13] It is believed that a symptom-limited, corridor walk may fall somewhere between the treadmill-walk test and the step test, in terms of producing exercise-induced oxyhemoglobin desaturation.[13]

A corridor walk is one method that has been employed to determine oxygen flow requirements during exercise.[13,15,16] Variation exists in the interpretation of oximetry values used to determine oxygen-flow requirements.[13,15,16] In general, COPD patients are questioned regarding their normal activity level, and attempts are made to titrate their oxygen flow so that they remain adequately oxygenated during their normal activities of daily living (ADLs). This retrospective analysis was conducted to determine whether pulse oximetry (S_{pO_2}) measurements,* taken during a 5-minute, symptom-limited corridor walk, accurately predict oxygen flow requirements for the standard nasal cannula (SNC) and reservoir nasal cannula (RNC) so that an average $S_{pO_2} \geq 91\%$ can be maintained in patients with hypoxemic COPD during the performance of simulated ADLs.

Methods

This research was approved by the hospital's Human Studies Subcommittee, and written, informed consent was obtained from the participants. Seventy-five COPD patients were contacted and screened for study participation. Among those contacted and screened, 43% did not meet the study's inclusion criteria and 29% met the study's inclusion criteria but refused to participate in the study. Twenty-one patients were enrolled in the investigation, and 15 patients successfully completed the study (2 patients were unable to finish the study due to exercise intolerance, and 4 patients elected to discontinue participation after starting the study). Baseline patient data were obtained (Table 1). Inclusion criteria were: (a) $P_{aO_2} \leq 55$ torr or 56-59 torr with evidence of cor pulmonale or polycythemia while upright and at rest and breathing room air; (b) $P_{aO_2} \geq 60$ torr with decline in average S_{pO_2} to $\leq 88\%$ during a 5-minute, symptom-limited corridor walk; (c) ratio of volume exhaled in the first second of a forced exhalation to forced vital capacity ($FEV_1/FVC\%$) < 70%; (d) no unstable medical or psychiatric illness; (e) no recent (within 4 weeks) infectious exacerbation of COPD as reflected by increase in quantity or change in color of sputum, elevated white blood count above 10,000 cells/mm^3, fever, or new pulmonary infiltrate; (f) no evidence of myocardial infarction within the previous 6 months.

*Suppliers of commercial products are listed in the Product Sources section at the end of the text.

Table 1. Mean (SD) Demographic and Clinical Data for Study Participants (n = 15) and Subjects Who Did Not Complete the Study (n = 6).

	Participants	Nonparticipants
Age (years)	69.2 (9.2)	69.0 (8.3)
Weight (kg)	81.3 (15.8)	88.1 (15.6)
Height (cm)	174.5 (6.6)	172.3 (9.0)
Years of smoking	40.5 (16.5)	35.0 (11.8)
Years since quitting smoking	8.9 (7.4)	7.3 (8.2)
FEV_1 (L)	1.0 (0.6)	0.9 (0.5)
FVC (L)	2.4 (0.9)	2.0 (0.7)
$FEV_1/FVC\%$	40.8 (10.1)	42.0 (13.0)
MVV (L/mm)*	39.9 (23.8)	28.6 (20.0)
D_{LCO} (mL/min/torr)†	9.3 (4.1)	5.6 (4.0)
P_{aO_2}‡	57.3 (4.2)	54.3 (3.4)
P_{aCO_2}‡	42.8 (8.2)	47.0 (7.3)
pH‡	7.4 (0.0)	7.4 (0.1)

$FEV_1/FVC\%$, MVV, D_{LCO} and P_{aO_2} were compared between groups; $p \geq 0.05$ for all comparisons. FEV_1 = forced exhalation volume in the first second. FVC = forced vital capacity. MVV = maximum voluntary ventilation. D_{LCO} = diffusing capacity for carbon monoxide.
*n = 14 for study participants; n = 5 for study nonparticipants. †n = 14 for participants; n = 3 for nonparticipants. ‡n = 13 for study participants; n = 6 for nonparticipants.

Prescreening Exercise Oximetry Protocol

The prescreening goal was to choose the SNC oxygen flow that achieved an average $S_{pO_2} \geq 91\%$ during a 5-minute, symptom-limited corridor-walk test (Table 2). The oximeter recorded S_{pO_2} every 6 seconds and then calculated the lowest S_{pO_2}, average S_{pO_2}, percent time $S_{pO_2} < 90\%$, and percent time $S_{pO_2} < 85\%$. Five minutes prior to testing and under the supervision of the study nurse, each patient self-administered 2 puffs of albuterol or metaproterenol via metered dose inhaler (MDI) to minimize bronchospasm as a cause of exercise-test intolerance. The oximeter probe was meticulously secured to the patient to prevent motion artifact. Testing was begun by having the patient walk at a self-selected pace on level ground, while using his currently prescribed SNC oxygen flow. If the average S_{pO_2} was $\geq 91\%$, that SNC oxygen flow was used in the study protocol. If the average S_{pO_2} was < 91%, the walk was repeated, while increases in the oxygen flow were made in increments of 0.5 L/min until an average $S_{pO_2} \geq 91\%$ was achieved or until the research nurse noted no further increase in S_{pO_2} with increased oxygen flow. All patients returned to their pre-exercise levels prior to subsequent corridor-walk tests.[16] Rest time ranged from 5 to 15 minutes. The 0.5 L/min increases in oxygen flow titration occurred over several separate exercises. The 0.5 L/min increment was used for reasons of cost containment and patient convenience. A pre- and postactivity heart rate and standing blood pressure (BP) were recorded for each walk test.

The procedure for evaluating the RNC began with a resting oximetry test with adjustments in oxygen flow made until

Table 2. Selection of Oxygen Flow (L/min) for Standard Nasal Cannula and Reservoir Nasal Cannula during 5-Minute Corridor Walk.

	Standard Nasal Cannula					Reservoir Nasal Cannula				
Subject	O_2 Flow (L/min)	Walk Time (min)	Lowest S_{pO_2} (%)	Mean S_{pO_2} (%)	Highest S_{pO_2} (%)	O_2 Flow (L/min)	Walk Time (min)	Lowest S_{pO_2} (%)	Mean S_{pO_2} (%)	Highest S_{pO_2} (%)
1	3.0	5.1	89	91	94	3.0	4.0	88	91	94
2	2.5	5.0	87	91	98	1.5	4.4	87	91	95
3	1.5	5.1	89	91	92	1.5	5.3	95	96	97
4	3.5	5.2	88	92	95	1.5	5.1	88	91	95
5	3.0	4.0	87	92	97	2.0	5.0	91	93	94
6	3.0	5.0	89	91	95	2.0	4.3	89	93	95
7	4.0	5.1	92	94	97	2.0	4.5	90	93	96
8	4.0	4.1	87	91	94	4.0	4.1	88	91	94
9	2.0	5.4	85	92	100	1.0	4.0	85	92	100
10	3.0	5.0	89	92	96	1.0	5.0	88	92	97
11	3.0	4.3	82	92	96	2.5	5.1	89	93	97
12	3.0	4.3	89	91	95	2.0	4.4	88	91	94
13	2.5	5.0	88	93	97	1.0	5.0	85	92	97
14	2.0	5.0	87	91	96	0.5	5.1	88	91	96
15	2.5	5.0	88	92	98	1.5	5.0	87	91	96
Mean (SD)	2.8 (0.7)	4.8 (0.4)	88 (2)	92 (1)	96 (2)	1.8 (0.9)	4.7 (0.5)	89 (2)	92 (1)	96 (2)

an average S_{pO_2} of $\geq 91\%$ was obtained. Patients then walked 5 minutes in the corridor, using their predetermined RNC oxygen flow. If that oxygen flow achieved an average $S_{pO_2} < 91\%$, the walk was repeated with the same titration procedure as used for SNC.

Study Protocol

Prior to initiating each exercise test, an arterial blood sample was obtained via radial artery puncture, while the patient was in a seated position with the pulse oximeter ear probe in place. Partial pressure of arterial oxygen (P_{aO_2}), oxyhemoglobin saturation (S_{aO_2}), and carboxyhemoglobin saturation (S_{aCO}) were measured. The S_{aO_2} and S_{pO_2} resting measurements were required to be within 2% in order to proceed with the testing protocol.

Measurements of heart rate, BP, S_{pO_2}, and ratings of perceived breathlessness and exertion were recorded before activity, during activity, and after activity for cycle and functional performance test circuit (FPTC) exercise. During cycle ergometry, for reasons of patient safety, electrocardiogram (ECG) heart rate (HR) was monitored visually. A telemetry system was used for ECG monitoring during the FPTC. ECG recording took place during the last 15 seconds of Minutes 10 and 15 of the pretest rest period and posttest recovery periods and during the last 15 seconds of each minute required by the subject to complete the specified physical activity. Blood pressure was measured by sphygmomanometry. The first and last Korotkoff sounds were recorded. Blood pressure was measured during the last 30 seconds of the pretest rest and posttest recovery periods, immediately following the completion of each physical activity, and during the last 30 seconds of the third minute of the recovery period.

Oximetry data were obtained as during prescreening. Ratings of perceived exertion and breathlessness were obtained using Borg's ratio scale, a standard method that has proved reliable and valid for measuring dyspnea and other subjective sensations in COPD patients.[17,18] Exertion and breathlessness ratings were taken before beginning and immediately after the subject finished a physical activity and at 1, 2, and 3 minutes of postactivity recovery. Subjects continued the exercises until (a) the subject completed the required task, (b) the subject was unable to maintain the designated work intensity, (c) volitional fatigue occurred, or (d) significant clinical signs or symptoms appeared during any physical activity.

Cycle Ergometry Protocol

Cycle exercise rather than treadmill exercise was employed because workload is more easily quantified during cycle exercise. Each subject performed cycle ergometry on 2 separate occasions, using SNC and RNC, with the oxygen flows determined during the 5-minute corridor-walk test. Beginning with pedaling at 0 watts (W), the work rate was increased by 10 W every 2 minutes until the subject indicated that exercise-limiting fatigue and/or dyspnea had occurred or 85% of the subject's predicted maximal heart rate (220 − age) was achieved.[19] Measurements were recorded during the last minute of each stage of bicycle exercise and for each of 3 minutes immediately following exercise.

FPTC Protocol

The 4-station FPTC was designed to mimic a variety of activities of daily living, including physical activities such as dressing, standing, walking, lifting, carrying, and ascending

and descending stairs. A 15-minute pretest quiet period was used to obtain the pre-exercise, stable baseline measurements. Subjects then proceeded through the stations in the same order, beginning with Station I. The FPTC test design is analogous to an intermittent exercise test in which a period of exercise is followed by a rest period of specified duration (in this case, 5 minutes). The FPTC was administered as described in Table 3 and shown in Figures 1 and 2.

Table 3. Protocol for Functional Performance Test Circuit

Station & Description	Instruction
I. Lift-push-carry (Arm & leg exercise)	(a) Begin seated. (b) Stand, &, in pace with a metronome, (c) Walk 15 feet. (d) Lift 21-pound grocery bag. (e) Place bag in cart. (f) Push Cart 60 feet. (g) Remove bag, and carry 45 feet. (h) Place bag on table. (i) Sit down.
II. Undress & dress (Arm, trunk, & leg exercise)	(a) Begin seated. (b) Remove shirt, pants, socks, & shoes. (c) Immediately, put on shirt, pants, socks, & shoes. (d) Sit down.
III. Arm work above shoulders & below knees (Arm, leg, & trunk exercise)	(a) Begin seated. (b) Stand &, in pace with a metronome, (c) Walk 15 feet. (d) Pick up 12-pound box. (e) Carry box 60 feet. (f) Place box on floor under 2 shelves. (g) Remove, 2 at a time, the six 2-pound sandbags from the box on the floor, and place them on the lower shelf. (h) Remove sandbags from the low shelf, 2 at a time, & place on the high shelf. (i) Continue transferring sandbags from shelf to shelf for 1 minute. (j) Walk 15 feet. (k) Sit down.
IV. Ascend & descend stairs (Leg exercise)	(a) Begin seated. (b) Stand &, in pace with a metronome, (c) Walk 5 feet. (d) At a stepping rate set by the metronome, ascend & descend 7 stairs continuously for 1 minute. (e) Sit down.

Data Analyses

Analysis of variance for repeated measures (ANOVA-r) was used to compare heart rate, BP, ratings of perceived breathlessness and exertion, and S_{pO_2}. A Student-Newman-Keuls test was used to make post hoc pairwise comparisons. Alpha was set at 0.05 for these analyses.

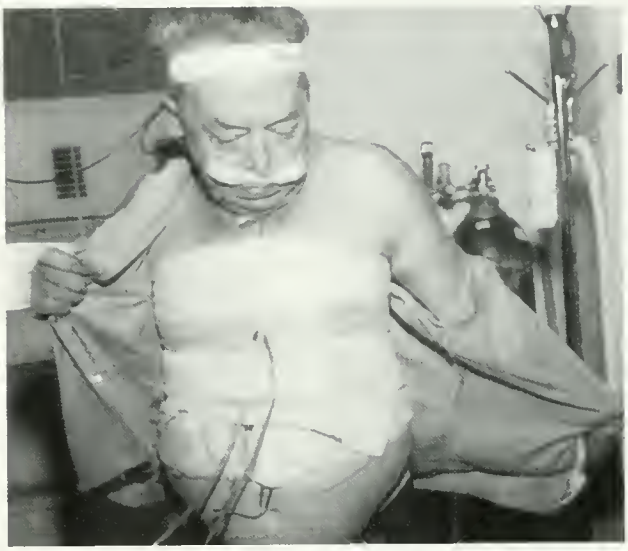

Fig. 1. Patient removing his shirt during undress-and-dress activity.

Fig. 2. Patient lifting sandbags onto a shelf during arm-work-above-shoulders-and-below-knees activity.

Results

There were no differences between the demographic and clinical data of patients who completed the study and patients who did not complete the study ($p \geq 0.05$, Table 1). Of those patients completing the study, 1 had moderate COPD ($FEV_1/FVC\%$ 60-69%), 1 had moderately severe COPD ($FEV_1/FVC\%$ 50-59%), 8 had severe COPD ($FEV_1/FVC\%$ 34-49%) and 5 had very severe COPD ($FEV_1/FVC\% < 34\%$). In the group of patients who failed to complete the study, 1 patient had moderate COPD, 1 patient had moderately severe COPD, 2 patients had severe COPD, and 2 patients had very severe COPD. Of the 15 patients who completed the study,

3 did not have a room-air resting $P_{aO_2} \leq 55$ torr or 56-59 torr with evidence of cor pulmonale or polycythemia, but they did have a decline in $S_{pO_2} \leq 88\%$ (average) during a 5-minute, symptom-limited, corridor walk. The time between the prescreening corridor walk and study completion ranged from 7-10 weeks.

The difference between S_{aO_2} and S_{pO_2} ($S_{aO_2} - S_{pO_2}$) was never > 0.40%. The results of data gathered during the prescreening phase of the study are given in Table 2. There were no significant differences in heart rate, BP, or in ratings of perceived breathlessness and exertion between the exercise conditions (Table 4). During RNC use, the differences in S_{pO_2} (Mean [SD]) between the WALK$^{:30}$ (90 [3]%) and both lift-push-carry (94 [3]%), and ascending and descending stairs (93 [3]%) were significant ($p < 0.01$ and 0.05, respectively). When SNC was used, the differences between WALK^{30} (90 [3]%), lift-push-carry (94 [2]%), undressing and dressing (Fig. 3) (92.1 [3.1]), and ascending and descending stairs (93.3 [2.8]) were significantly different.

The oxygen flow selected for SNC and RNC was based upon an average $S_{pO_2} \geq 91\%$ during the 5-minute, symptom-limited corridor walk (Table 2). Average oxygen flow [SD] required for exercise was 1.8 [0.9] L/min for the RNC and 2.8 [0.7] L/min for SNC. While using SNC, 8 patients (53%) had their lowest S_{pO_2} during the last 30 seconds of the 5-minute corridor walk, 1 patient (7%) during the cycle test, 2 patients (13%) during undressing and dressing activity, 3 patients (20%) during arm work above shoulder level, and 1 patient (7%) during stair activity. While using RNC, 9 patients (60%) had their lowest S_{pO_2} during the last 30 seconds of the 5-minute corridor walk, 1 patient (7%) during arm work above shoulder level, 1 patient (7%) during stair activity, 1 patient (7%) during lift-push-carry activity, and 3 patients (20%) during the cycle test (Table 5). One explanation for the 3 patients who had their lowest S_{pO_2} during the cycle test is that they exercised longer using the RNC (9.6 [4.9] min) than they did during SNC use (7.5 [4.2] min).

Discussion

A variety of protocols are used by health-care providers to evaluate exercise-induced oxyhemoglobin desaturation in COPD patients. Frequently, pulse oximetry is used to determine a patient's exercise oxygen prescription.[15,16] However, studies regarding the validity and reliability of pulse oximetry have shown that the pulse oximetry values obtained during exercise may be dangerously misleading and that they should be interpreted with caution.[20-24] A substantial difference in bias and precision among different pulse oximeters has been found,[23] the most common problems being underestimation of saturation and failing precision.[23] In hypoxemic COPD patients, the lower the S_{aO_2}, the larger the overestimation of S_{aO_2} by pulse oximetry.[25] The American Association for Respiratory Care recommends that the accuracy of pulse oximetry be verified by comparison to measured arterial oxygen saturation at least once during the performance of exercise testing that is being done to evaluate patients for the presence of exercise-induced hypoxemia.[16]

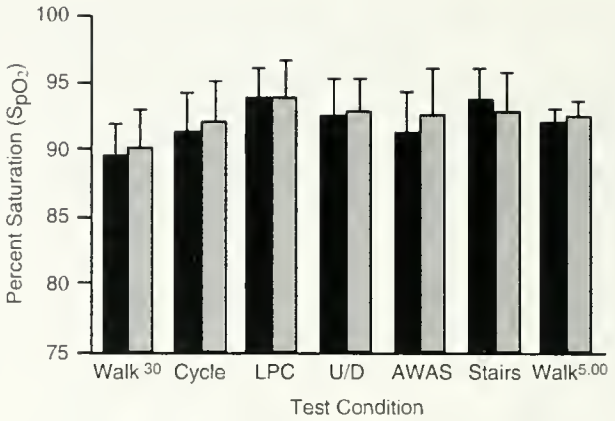

Fig. 3. Oxyhemoglobin saturation measured by pulse oximetry (S_{pO_2}) as subjects used a standard nasal cannula (■) and reservoir nasal cannula (▒) while completing bicycle ergometry, functional performance test circuit composed of 4 stations: undress and dress (U/D), lift-push-carry (LPC), arm work above shoulders (AWAS), ascending and descending stairs (Stairs), a 5-minute corridor walk with S_{pO_2} determined as the average of measures during 5 minutes and during the last 30 seconds of walking.

Table 4 Subjects' (n = 15) Mean (SD) Heart Rate (Last 30 s; End Exercise Corridor Walk), Systolic and Diastolic Blood Pressure (BP, End Exercise), Perceived Breathlessness, and Total Body Exertion (Last 15 s) for the Standard Nasal Cannula (SNC) and Reservoir Nasal Cannula (RNC).

	Post Corridor Walk		Cycle Ergometer		FPTC Stations* I		II		III		IV	
	SNC	RNC	SNC	RNC	SNC	RNC	SNC	RNC	SNC	RNC	SNC	RNC
Heart rate	106 (15)	108 (13)	125 (13)	123 (12)	107 (7)	108 (9)	108 (14)	109 (10)	109 (9)	110 (10)	110 (8)	110 (11)
Systolic BP	133 (23)	135 (20)	157 (29)	165 (24)	149 (24)	144 (28)	152 (26)	154 (25)	155 (26)	157 (26)	155 (28)	158 (26)
Diastolic BP	84 (9)	83 (7)	93 (8)	90 (8)	79 (10)	80 (10)	80 (10)	80 (11)	78 (7)	82 (8)	82 (12)	84 (10)
Breathlessness	—	—	5 (3)	6 (3)	3 (2)	2 (2)	3 (2)	3 (3)	3 (2)	3 (2)	4 (3)	4 (2)
Exertion	—	—	5 (3)	5 (4)	2 (2)	2 (2)	3 (2)	3 (3)	3 (2)	2 (2)	3 (3)	3 (3)

*FPTC = functional performance test circuit; no comparisons were significant ($p \geq 0.05$). —, measurement not taken.

Table 5. For Standard Nasal Cannula and Reservoir Nasal Cannula Mean S_{pO_2} (%) during Last 30 Seconds of the 5-Minute Corridor Walk, Cycling, and Functional Performance Test Circuit.

	Standard Nasal Cannula						Reservoir Nasal Cannula					
Subject	Walk	Cycle	LPC	U/D	AWAS	Stairs	Walk	Cycle	LPC	U/D	AWAS	Stairs
1	90	96	95	94	92	95	89	94	97	96	92	96
2	87	89	96	92	92	92	88	92	93	93	94	94
3	91	87	90	86	91	90	96	89	93	93	90	93
4	93	90	92	90	92	93	88	91	94	94	97	96
5	87	88	95	91	95	96	92	93	96	94	96	94
6	90	94	96	92	88	97	93	93	91	93	89	98
7	93	92	94	95	90	95	93	91	93	92	92	91
8	87	94	91	90	94	94	90	94	93	92	91	94
9	93	92	98	98	98	92	92	97	99	98	100	92
10	91	87	89	88	86	89	88	90	87	88	88	90
11	83	91	94	96	88	97	91	95	97	93	95	92
12	89	92	94	95	89	96	89	94	92	92	91	94
13	89	92	92	90	90	95	86	84	94	90	90	90
14	88	91	92	92	93	88	88	89	92	87	89	86
15	88	95	93	92	90	91	87	93	93	92	90	91

Walk = 5-minute, symptom-limited, corridor walk; Cycle = submaximal, symptom-limited, bicycle-ergometer test. LPC = lift-push-carry activities; U/D = undressing and dressing activity; AWAS = Arm work above shoulder level, 1-minute duration; Stairs = ascending and descending stairs, 1-minute duration; NOTE: Some subjects rode longer on the cycle than others. Not all patients completed 5 minutes on the corridor walk.

In the present investigation, the average S_{pO_2} measured during the last 30 seconds of the corridor walk produced a level of desaturation that was significantly greater than that during the lift-push-carry, ascending and descending stairs, and undressing and dressing. There was no significant difference between WALK:[30] and bicycle ergometry. However, the means differed by 2% during SNC use and 1.8% during RNC use. Although the means for the WALK[5:00] and WALK:[30] were not statistically different from each other, the individual S_{pO_2} measures may have some relevance to clinical practice. The subjects' mean S_{pO_2} for the WALK[5:00] are all $\geq 91\%$ for both the SNC and RNC (Table 2). Whereas, during WALK:[30] the mean S_{pO_2} was $< 91\%$ in 67% and 60% of the subject trials during SNC and RNC use (Table 5). Based on these observations, it appears that SNC and RNC oxygen flow prescriptions intended to maintain an $S_{pO_2} \geq 91\%$ during physical exertion in persons with hypoxemic COPD may be determined during a 5-minute corridor walk where only the oximetry measures taken during the last 30 seconds of exercise are used to set the patient's oxygen therapy flow. However, the lowest S_{pO_2} for several patients was recorded during activities other than the corridor walk.

Initial and ongoing assessment of arterial oxyhemoglobin saturation during rest and exercise is necessary to ensure an oxygen prescription adequate to support ADLs for hypoxemic COPD patients. The results of this retrospective analysis underscore the need for further research to standardize and improve the clinical methods used for exercise oxygen prescription. The potential benefits are better patient care and cost reduction.

PRODUCT SOURCES

Pulse Oximeter:
Model 3760, Ohmeda, Louisville CO

Blood-Gas Analyzer:
ABL 520 Radiometer, Medtron, Des Plaines IL

Pulmonary Function and D_{LCO}:
PK Morgan Instruments Inc, Andover MA

Stress Monitor & Exercise Cycle:
Quinton Instrument Company, Seattle WA

Telemetry System:
Escort 100T/200T, Medical Data Electronics, Arleta CA

Statistical Analysis Package:
StatView 4.01, Abacus Concept Inc, Berkeley CA

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REFERENCES

1. Vyas MN, Banister EW, Morton JW, Grzybowski S. Response to exercise in patients with chronic airway obstruction. II. Effects of breathing 40 percent oxygen. Am Rev Respir Dis 1971;103(3):401-412.

2. King AJ, Cooke NJ, Leitch AG, Flenley DC. The effects of 30 percent oxygen on the respiratory response to treadmill exercise in chronic respiratory failure. Clin Sci 1973;44(2):151-162.
3. Bradley BL, Garner AE, Billiu D, Mestas JM, Forman J. Oxygen-assisted exercise in chronic obstructive lung disease. The effect on exercise capacity and arterial blood gas tensions. Am Rev Respir Dis 1978;118(2):239-243.
4. Davidson AC, Leach R, George RJ, Geddes DM. Supplemental oxygen and exercise ability in chronic obstructive airways disease. Thorax 1988;43(12):965-971.
5. Dean NC, Brown JK, Himelman RB, Doherty JJ, Gold WM, Stulbarg MS. Oxygen may improve dyspnea and endurance in patients with chronic obstructive pulmonary disease and only mild hypoxemia. Am Rev Respir Dis 1992;146(4):941-945.
6. Kawakami Y, Terai F, Yamamoto H, Murao M. Exercise and oxygen inhalation in relation to prognosis of chronic obstructive pulmonary disease. Chest 1982;81(2):182-188.
7. Scano G, Van Meerhaeghe A, Willeput R, Vachandez JP, Sergysels R. Effect of oxygen on breathing during exercise in patients with chronic obstructive lung disease. Eur J Respir Dis 1982;63(1):23-30.
8. Keller CA, Shepard JW Jr, Chun DS, Vasquez P, Dolan GF. Pulmonary hypertension in chronic obstructive pulmonary disease. Multivariate analysis. Chest 1986;90(2):185-192.
9. Himelman RB, Abbot JA, Lee E, Schiller NB, Dean NC, Stulbarg MS. Doppler echocardiography and ultrafast cine computed tomography during dynamic exercise in chronic parenchymal pulmonary disease. Am J Cardiol 1989;64(8):528-533.
10. Hunt JM, Copland J, McDonald CF, Barter CE, Rochford PD, Pierce RJ. Cardiopulmonary response to oxygen therapy in hypoxemic chronic airflow obstruction. Thorax 1989;44(11):930-936.
11. Lane R, Cockcroft A, Adams L, Guz A. Arterial oxygen saturation and breathlessness in patients with chronic obstructive airways disease. Clin Sci 1987;72(6):693-698.
12. Soffer M, Tashkin DP, Shapiro BJ, Littner M, Harvey E, Farr S. Conservation of oxygen supply using a reservoir nasal cannula in hypoxemic patients at rest and during exercise. Chest 1985;88(5):663-668.
13. Mak VH, Bugler JR, Roberts CM, Spiro SG. Effect of arterial oxygen desaturation on six minute walk distance, perceived effort, and perceived breathlessness in patients with airflow limitation. Thorax 1993;48(1):33-38.
14. Cockcroft A, Beaumont A, Adams L, Guy A. Arterial oxygen desaturation during treadmill and bicycle exercise in patients with chronic obstructive airways disease. Clin Sci 1985;68(3):327-332.
15. Nelson CM, Murphy EM, Bradley JK, Durie RH. Clinical use of pulse oximetry to determine oxygen prescriptions for patients with hypoxemia. Respir Care 1986;31(8):673-680.
16. American Association for Respiratory Care. Clinical practice guideline: exercise testing for evaluation of hypoxemia and/or desaturation. Respir Care 1992;37(8):907-912.
17. Borg GAV. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 1982;14(5):377-381.
18. Silverman M, Barry J, Hellerstein H, Janos J, Kelsen S. Variability of the perceived sense of effort in breathing during exercise in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1988;137(1): 206-209.
19. Brown SE, Fischer CE, Stansbury DW, Light RW. Reproducibility of VO_{2max} in patients with chronic air-flow obstruction. Am Rev Respir Dis 1985;131(3):435-438.
20. Escourrou PJ, Delaperche MF, Visseaux A. Reliability of pulse oximetry during exercise in pulmonary patients. Chest 1990;97(3):635-638.
21. Hansen JE, Casaburi R. Validity of ear oximetry in clinical exercise testing. Chest 1987;91(3):333-337.
22. Orenstein DM, Curtis SE, Nixon PA, Hartigan ER. Accuracy of three pulse oximeters during exercise and hypoxemia in patients with cystic fibrosis. Chest 1993;104(4):1187-1190.
23. Severinghaus JW, Naifeh KH, Koh SO. Errors in 14 pulse oximeters during profound hypoxia. J Clin Monit 1989;5(2):72-81.
24. Ries AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. Am Rev Respir Dis 1985;132(3):685-689.
25. Hannhart B, Michalski H, Delorme N, Chapparo G, Polu JM. Reliability of six pulse oximeters in chronic obstructive pulmonary disease. Chest 1991;99(4):842-846.

Tracheostomy in the 1990s: Current Clinical Practice in an Urban University Teaching Hospital

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BACKGROUND: Tracheotomy is a commonly performed procedure in hospitals and may be performed for various reasons by one of several services. Because of the absence of both a defined institution-wide procedure for assessment and recent literature to describe current tracheostomy management, we conducted this retrospective study. METHODS: We reviewed the medical records of all patients for the fiscal year 1992-93 coded as having tracheostomy status prior to admission or having tracheotomy performed during the admission. We attempted to answer (1) Why were tracheotomies performed? (2) Who performed/managed the tracheotomies? (3) What postoperative care was given? RESULTS: Of the 109 patients, 74% were men 13 to 83 years of age. Patients underwent tracheotomy for facial injuries (21%), prolonged ventilation (21%), closed head injuries (20%), other intracranial processes (16%), other head and neck surgery (6%), spinal cord injuries (5%), epiglottitis (3%), and 8% underwent tracheotomy for reasons other than these. Thirty-seven percent were tracked prior to this admission. Of the 93 admissions, 63% had tracheotomy performed during the current admission by General Surgery (27%), Otolaryngology Head and Neck Surgery (46%), Neurosurgery (22%), Oral and Maxillofacial Surgery (2%), Burns-Plastic Surgery (3%). Of the patients with tracheostomies 67% had orders and 85% had progress notes pertaining to tracheostomy management. Of the 147 admissions, 100 (68%) had Respiratory Care notes regarding tracheostomy. CONCLUSION: Because a number of services with various training backgrounds participate in the care of patients with tracheostomy and because variation exists, a multidisciplinary tracheostomy team appears desirable in a hospital such as ours. [Respir Care 1996;41(1):37-42]

Introduction

Tracheostomy care is a long-established and important aspect of respiratory care.[1-6] In general hospitals, tracheotomy may be performed for a wide range of reasons[7-13] in adults,[14-16] children,[17-20] or neonates.[22-24] Intraoperative and perioperative complications of the tracheotomy procedure[25-27] and acute and long-term complications occurring in patients with tracheostomies[28-32] are numerous and well documented. Although regimens for tracheostomy care are widely available,[1-4,6,33,34] none has gained universal adoption, and variations likely exist among institutions and regions.

Harborview Medical Center is a university-operated, municipal teaching hospital and Level-1 Trauma Center. Included

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in its 320 beds are 38 intensive-care-unit (ICU), 187 acute-care, and 26 rehabilitation beds. Although a substantial proportion of the patients have suffered trauma and other emergencies, the hospital also serves the needs of a varied local and regional community. Except for certain emergency admissions to the surgical services, infants and children are cared for as inpatients at a separate but affiliated hospital.

Tracheotomy is performed in the operating room by surgeons from the Departments of General Surgery, Otolaryngology/Head-Neck Surgery (OTO/HNS), Neurosurgery, Oral and Maxillofacial Surgery (OMFS), and Burn Surgery. In patients on nonsurgical services, the procedure is most often performed by the OTO/HNS service in consultation with the attending, or primary, physician. The tracheostomy is managed either by the attending physicians or by the consulting surgeons who performed the procedure. There is no fixed policy for the care of patients with pre-existing tracheostomies; they are cared for by the attending service admitting them. Orders pertaining to tracheostomy care are generally written by the managing physicians. Although members of the Respiratory Care Department often participate in tracheostomy management, during the study period there was no requirement or fixed protocol for their involvement. Members of the Department of Speech Pathology are often asked to perform swallowing evaluations as part of the assessment for decannulation, but this also is not standardized.

To better characterize current tracheostomy care in this setting, we reviewed the medical records of all patients who underwent tracheotomy or were recorded as having a tracheostomy at our institution, during a recent 1-year period. We sought to determine how many such patients were cared for in our hospital, what their primary and tracheostomy-related diagnoses were, which services performed the procedure and managed the tracheostomy afterwards, and what the clinical outcomes of these admissions were. We also evaluated the adequacy of documentation of orders and progress notes related to the tracheostomy. In patients who underwent decannulation during the hospitalization, we looked for documentation of involvement by respiratory care, speech pathology, and other consulting services in this process.

Materials & Methods

All hospital inpatient admissions between July 1, 1992, and June 30, 1993, were considered for the study. Patients were identified, through discharge diagnosis and procedure codes, as either having undergone a tracheotomy during the hospitalization or having a tracheostomy at the time of admission. The charts were reviewed by two of the authors (CLK and CRS), and data were recorded. Data were collected under the general headings of patient demographics, admitting diagnosis, medical information and ICU care, tracheostomy data, and data from swallow evaluation or other speech-pathology assessments.

Data were then transcribed to computer sheets and analyzed. Data were entered into a relational database (R:Base, Microrim, Bellevue WA) and summary statistics generated using commercially available statistical-analysis software (SAS Institute, Cary NC).

Results

Patient Demographics, Diagnoses, & Outcomes

Total inpatient admissions during the study period were 16,491. We coded 120 patients as either undergoing tracheotomy or having tracheostomy status. Of the 120 charts, 8 had been miscoded (no evidence of tracheostomy in the record), and 3 were unavailable for review. Thus, the study population was comprised of 109 patients, with a total of 147 admissions, during the period of this investigation (Table 1).

Table 1. The Distribution of Patients in Our Sample According to the Number of Admissions during the Study Period.

Number of Admissions	n
1	90
2	11
3	3
4	1
5	3
6	0
7	1

Of the 109 patients, 81 (74%) were men and 28 (26%) were women. Their ages ranged from 13 to 83 years (mean [SD] = 44.5 [19.8] years). Admitting services and diagnoses for the 147 admissions are shown in Table 2.

Table 2. Admitting Service and Patient Diagnosis (n = 147 Admissions)

Admitting Service	Admissions(n)	%n
General Surgery	42	28
Medicine	37	25
Neurosurgery	29	20
Neurology	16	11
OTO/HNS*	12	8
Rehabilitation Medicine	7	5
Other	4	3
Admitting Diagnosis	**Admissions (n)**	**%n**
Trauma	62	42
Strokes & Other CNS* Events	18	12
Pneumonia	16	11
Malignancies	13	9
Other	38	26

*OTO/HNS = otolaryngology/head-neck surgery; CNS = central nervous system.

Length of hospital stay for the 147 admissions ranged from 1 to 136 days (mean [SD] = 32.4 [30.4] days, median 24 days). Outcomes of hospitalization are shown in Table 3.

Table 3. Outcomes of Hospitalizations (n = 147 Admissions)

Discharged to	Admissions (n)	%n
Subacute Care Center	57	39
Home	42	29
Another Acute Care Hospital	18	12
Death in Hospital	16	10
Nursing Home	7	5
Another Institution	7	5

Patients were managed in the ICU on at least one occasion during 100 of the 147 admissions (68%). In 69 of the 147 admissions (47%), the patient was receiving ventilatory support at the time of admission to the hospital, and patients underwent mechanical ventilation at some time during their stay in 92 admissions (63%). Patients met diagnostic criteria for the acute respiratory distress syndrome[35] during 13 admissions (9%). Of the 93 patients who underwent tracheotomy in our institution, 63 (68%) were receiving ventilatory support at the time of the procedure.

In 64 of the 93 admissions (68%) in which mechanical ventilation was required, the patient could not be weaned from ventilatory support during that hospitalization. Ultimate disposition for these 64 admissions is illustrated in Table 4.

Table 4. Ultimate Disposition in 64 Patients Who Weaned from Ventilatory Support

Disposition	Admission (n)	%n
Home	24	37
Long-Term Subacute-Care Facility	21	33
Death in Hospital	9	14
Another Hospital	5	8
Nursing Home	5	8

Tracheotomy Indications & Circumstances

Tracheotomies were performed during 93 (63%) of the 147 admissions, and a tracheostomy was already in place on 54 admissions. For the 93 procedures, the primary indications varied considerably, as shown in Table 5. About three fourths of the patients had the procedure because of the need for prolonged ventilatory support, extensive facial injuries, closed-head injury, or other severe, central-nervous-system condition—all in nearly equal proportions—reflecting our institution's status as a Level-1 trauma center. Elective head and neck surgery for malignancy and other disorders accounted for only 6 procedures. Acute spinal-cord injury requiring ventilatory support was the indication for tracheotomy in 5 instances. Three patients underwent tracheotomy because of acute epiglottitis and 7 because of one of a variety of other disorders.

Table 5. Indications for Tracheotomy (n = 147)

Diagnosis	%n
Facial Fracture	21
Prolonged Ventilation	21
Closed Head Injury	20
Other CNS* Event	16
Other	8
Elective Head and Neck Surgery	6
Spinal Cord Injury	5
Epiglottitis	3

*Central nervous system

All but 2 of the 93 tracheotomies done in our institution were performed in the operating room. One was performed in the emergency department, and one in the intensive care unit. One emergency tracheotomy was performed in the field prior to arrival at our institution (with subsequent operative revision). On six occasions patients were transferred to our facility having undergone tracheotomy at the referring hospital. When tracheotomy was carried out at our hospital, it was performed by OTO/HNS in 43 of 93 instances (46%), by general surgery in 25 (27%), by neurosurgery in 20 (22%), by burn surgery in 3 (3%), and by OMFS in 2 (2%).

Tracheostomy Management & Documentation

Overall, 98 of 147 charts (67%) contained physician orders, and 124 of 147 (84%) had progress notes pertaining to the tracheostomy (Tables 6 & 7). A service other than the patient's primary managing service was consulted about the tracheostomy (including consultations for performance of the procedure) on 53 of 147 admissions (36%).

Table 6. Orders Pertaining to Tracheostomy Management, by Service

Service	Admissions (n)	%n
OTO/HNS*	37/43	86
General Surgery	18/25	72
Neurosurgery	14/20	70

*Otolaryngology/head-neck surgery

Table 7. Progress Notes Pertaining to Tracheostomy, by Service

Service	Admissions (n)	%n
OTO/HNS*	42/43	98
General surgery	21/25	84
Neurosurgery	17/20	85

*Otolaryngology/head-neck surgery

Charts from 100 of 147 admissions (68%) contained notes by a respiratory care practitioner. Cuff volume was mentioned in 81 of these notes (81%), and cuff pressure in 88 (88%). When the tracheotomy had been performed during the current admission, the first Respiratory Care Department note appeared 3.2 [5.5] days (mean [SD]) after the procedure was performed.

Decannulation

The patient was successfully decannulated on 33 of 147 admissions (22%). Decannulation was accomplished by members of the Respiratory Care Department in 13 cases, by the managing physicians in 13, and by a consulting service (generally OTO/HNS) in 6; we were unable to tell in 1 instance who decannulated the patient.

Swallowing evaluations were made during 60 admissions (41%), and the patient was considered to have successfully 'passed' the evaluation in 26 instances (43%). Written recommendations of the speech pathologist were followed by the primary service in 57 of the 60 instances.

Discussion

In our university teaching hospital and Level-1 trauma center, tracheotomy is performed for a wide variety of indications, and the operation is performed by surgeons on 5 different services. Usually, general surgeons perform tracheotomies for their patients, as is also the case with the burn surgery and OMFS services. The Medicine, Neurology, and Neurosurgery services generally consult OTO/HNS for performance of the operation, and a substantial number of patients are also admitted to this service. A tracheostomy was already present prior to coming to our hospital in about one fourth of inpatient admissions involving tracheostomy, and patients with tracheostomies are admitted to every clinical service. Our patients ranged widely in age with a mean of 44 years, and the majority of them (74%) were men.

The largest single group of patients in this study was made up of those admitted for trauma, as reflects the nature of our institution. However, there is no set policy to dictate a tracheostomy for patients who have injury of a certain severity, as has been advocated by some,[36] and the procedure was performed when dictated by the judgment of the individual attending trauma surgeon.

There is little recent information in the literature on the spectrum of primary diagnoses in patients with tracheostomies or the indications for performing tracheotomy in a general hospital population. Previous studies have tended to focus either on aspects of the surgical procedure itself,[25,26,37] on complications,[14,27,29,31,32,38-40] or on tracheostomy in specific patient populations, such as those with trauma,[15,36] head injury,[10,32,40] spinal cord injury,[12] burns,[13] or Guillain-Barré syndrome.[11]

The nature of primary diagnoses encountered in children with tracheostomy seems to be different from that seen in our adult patients.[18,20,21] A review of 319 children who underwent tracheotomy at the Hospital for Sick Children in Toronto over a 10-year period[19] revealed primary diagnoses of airway abnormalities in 41%, craniofacial abnormalities in 27%, central nervous system disease in 18%, and infectious diseases in 14%. Most of the patients in this series (70%) had airway obstruction as the direct indication for tracheotomy; facilitation of tracheobronchial toilet was the indication in 20%, and prolonged mechanical ventilation in 10%.[19] In another series of 108 pediatric tracheostomies, 85 of them in infants ≤1 year of age, Simma et al[21] reported the indications for the procedure to be acquired subglottic stenosis in 31%, bilateral vocal cord paralysis in 22%, congenital airway malformations in 22%, and tumors in 11%.

In the only other recent study to examine tracheostomy practice in adult patients in a general hospital setting, Zeitouni and Kost reviewed 281 patients with tracheostomies at 2 large teaching hospitals in Montreal.[16] The average age of their patients was 60 years, and 64% were men. Tracheotomy was performed by OTO/HNS in 57% of patients, by general surgery in 24%, by cardiovascular surgery in 9%, by neurosurgery in 5%, by plastic surgery in 2%, and by OMFS in 0.35%. In comparison with our results, OTO/HNS did somewhat more, general surgery the same number, and neurosurgery fewer procedures; there is no cardiovascular surgery service in our institution.

Zeitouni and Kost did not give the primary diagnoses of their patients or the indications for tracheotomy. However, 56% of the patients were in the ICU, and the tracheotomy was generally performed "to aid in pulmonary toilet and weaning,"[16] implying that the need for prolonged ventilatory support was a major factor in the decision for tracheotomy. Ventilated patients had been intubated for 1-55 days (mean 18 days) at the time of tracheotomy. In the entire series, the procedure was performed electively in 88% of cases.

The need for prolonged mechanical ventilation, which was the only indication for tracheotomy in one reported series,[14] was the reason for performing the procedure in only 21% of our cases. This most likely reflects both the acute-care character of our institution and the prevailing practice of maintaining translaryngeal intubation for as long as 6 weeks, and even longer, in patients for whom extubation is a reasonable expectation within another 7-10 days.

There are several potential limitations to our study. Data gathered retrospectively very likely underestimate the true incidence of such complications as pneumonia, hemoptysis, and aspiration of gastric contents, and, for this reason, we abandoned our initial plan to include complications in our study results. After a preliminary review of 10 charts by 2 of us to ensure that data were recorded in the same manner, every subsequent chart was reviewed by only one investigator, thus, mistakes or subjective judgments by that single individual could have affected the results.

Although we did not document any differences in complications related to which hospital service cared for the patient with a tracheostomy, the large number of specialties and individual caregivers raises the possibility that not all of them have equivalent expertise in tracheostomy management. Table 8 demonstrates the actual and recommended incidence of notes in the medical records pertaining to tracheostomy management. These data again point to the need for a consistent mechanism to manage all patients with tracheostomies. Tracheostomy care has long been considered a fundamental skill for respiratory care practitioners.[1-3,41] The respiratory care department would thus seem a logical, unifying resource in the care of all tracheostomized patients, regardless of their primary diagnoses or the service responsible for their overall management.

Table 8. Actual and Recommended Incidences of Notes in the Medical Record Pertaining To Establishing and Maintaining Tracheostomies

Variable	Actual	Recommended
Cuff Volume Noted	68%	100%
Cuff Pressure Noted	88%	100%
MD Order Present	67%	100%
Progress Notes	84%	100%
First RCP Note	3.2 [5.5] days*	by 24 hours

*mean [SD]

The knowledge and skills of otolaryngologists, who deal more with upper-airway problems than do any other medical specialists, should be available to patients with tracheostomies if needed, as should access to pulmonary- and critical-care physicians and anesthesiologists. In addition, because of the frequency and importance of swallowing problems in patients with tracheostomies,[42] and the importance of adequate swallowing function in successful decannulation,[41] there is a natural role for speech pathologists in tracheostomy management. A care map or clinical pathway that included a multidisciplinary 'trach team' to oversee tracheostomy management and provide a resource for preventing and dealing with problems, coordinated through the respiratory care department, would thus appear to be desirable in a hospital such as ours.

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REFERENCES

1. Plevak DJ, Ward JJ. Airway management. In: Burton GB, Hodgkin JE, Ward JJ, editors: Respiratory care: a guide to clinical practice. Philadelphia: JB Lippincott, 1991:449-504.
2. Bishop MJ, Ritz RH. Airway management. In: Pierson DJ, Kacmarek RM, editors: Foundations of respiratory care. New York: Churchill Livingstone, 1992:825-841.
3. Shapiro BA, Harrison RA, Kacmarek RM, Cane RD. Tracheostomy. In: Clinical application of respiratory care, 3rd ed. Chicago: Year Book Medical Publisher Inc, 1985:242-264.
4. Heffner JE, Miller KS, Sahn SA. Tracheostomy in the intensive care unit. Part 1: indications, technique, management. Chest 1986;90(2): 269-274.
5. Heffner JE. Timing of tracheotomy in mechanically ventilated patients. Am Rev Respir Dis 1993;147(3):768-771.
6. Godwin JE, Heffner JE. Special critical care considerations in tracheostomy management. Clin Chest Med 1991;12(3):573-583.
7. Heffner JE. Medical indications for tracheotomy. Chest 1989;96 (1):186-190.
8. Marsh HM, Gillespie DJ, Baumgartner AE. Timing of tracheostomy in the critically ill patient. Chest 1989;96(1):190-193.
9. Lewis RJ. Tracheostomies. Indications, timing, and complications. Clin Chest Med 1992;13(1):137-149.
10. D'Amelio LF, Hammond JS, Spain DA, Sutyak JP. Tracheostomy and percutaneous endoscopic gastrostomy in the management of the head-injured trauma patient. Am Surg 1994;60(3):180-185.
11. Freezer NJ, Robertson CF. Tracheostomy in children with Guillain-Barré syndrome. Crit Care Med 1990;18(11):1236-1238.
12. Biering-Sorensen M, Biering-Sorensen F. Tracheostomy in spinal cord injured: frequency and follow up. Paraplegia 1992;30(9):656-660.
13. Jones WG, Madden M, Finkelstein J, Yurt RW, Goodwin CW. Tracheostomies in burn patients. Ann Surg 1989;209(4):471-474.
14. Gunawardana RH. Experience with tracheostomy in medical intensive care patients. Postgrad Med J 1992;68(799):338-341.
15. Rodriguez JL, Steinberg SM, Luchetti FA, Gibbons KJ, Taheri PA, Flint LM. Early tracheostomy for primary airway management in the surgical critical care setting. Surgery 1990;108(4):655-659.
16. Zeitouni AG, Kost KM. Tracheostomy: a retrospective review of 281 cases. J Otolaryngol 1994;23(1):61-66.
17. Puhakka HJ, Kero P, Valli P, Iisalo E. Tracheostomy in pediatric patients. Acta Paediatr 1992;81(3):231-234.
18. Duncan BW, Howell LJ, deLorimier AA, Adzick NS, Harrison MR. Tracheostomy in children with emphasis on home care. J Pediatr Surg 1992;27(4):432-435.
19. Crysdale WS, Feldman RI, Naito K. Tracheotomies: a 10-year experience in 319 children. Ann Otol Rhinol Laryngol 1988;97(5, Part 1):439-443.
20. Freezer NJ, Beasley SW, Robertson CF. Tracheostomy. Arch Dis Child 1990;65(1):123-126.
21. Simma B, Spehler D, Burger R, Uehlinger J, Ghelfi D, Dangel P, Hof E, Fanconi S. Tracheostomy in children. Eur J Pediatr 1994; 153(4):291-296.
22. Schlessel JS, Harper RG, Rappa H, Kenigsberg K, Khanna S. Tracheostomy: acute and long-term mortality and morbidity in very low birth weight premature infants. J Pediatr Surg 1993;28(7): 873-876.
23. Singer LT, Hill BP, Orlowski JP, Doershuk CF. Medical and social factors as predictors of outcome in infant tracheostomy. Pediatr Pulmonol 1991;11(3):243-248.
24. Zeitouni A, Manoukian J. Tracheotomy in the first year of life. J Otolaryngol 1993;22(16):431-434.
25. Stock MC, Woodward CG, Shapiro BA, Cane RD, Lewis V, Pecaro B. Perioperative complications of elective tracheostomy in critically ill patients. Crit Care Med 1986;14(10):861-863.
26. Waldron J, Padgham ND, Hurley SE. Complications of emergency and elective tracheostomy: a retrospective study of 150 consecutive cases. Ann R Coll Surg Engl 1990;72(4):218-220.
27. Myers EN, Carrau RL. Early complications of tracheotomy. Incidence and management. Clin Chest Med 1991;12(3):589-595.

28. Wood DE, Mathisen DJ. Late complications of tracheotomy. Clin Chest Med 1991;12(3):597-609.
29. Elpern EH, Scott MG, Petro L, Ries MH. Pulmonary aspiration in mechanically ventilated patients with tracheostomies. Chest 1994;105 (2):563-566.
30. Heffner JE, Miller KS, Sahn SA. Tracheostomy in the intensive care unit. Part 2: complications. Chest 1986;90(3):430-436.
31. Astrachan DI, Kirchner JC, Goodwin WJ Jr. Prolonged intubation vs tracheotomy: complications, practical and psychological considerations. Laryngoscope 1988;98(11):1165-1169.
32. Citta-Pietrolungo TJ, Alexander MA, Cook SP, Padman R. Complications of tracheostomy and decannulation in pediatric and young patients with traumatic brain injury. Arch Phys Med Rehabil 1993; 74(9):905-909.
33. Mason J, Murty GE, Foster H, Bradley PJ. Tracheostomy self-care: the Nottingham system. J Laryngol Otol 1992;106(8):723-724.
34. Fitton CM. Nursing management of the child with a tracheotomy. Pediatr Clin North Am 1994;41(3):513-523.
35. Bernard GR, Artigas A, Brigham KL, Carlet J, Falke K, Hudson L, et al. The American-European consensus conference on ARDS. Definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med 1994;149(3, Part 1):818-824.
36. Lesnik I, Rappaport W, Fulginiti J, Witzke D. The role of early tracheostomy in blunt, multiple organ trauma. Am Surg 1992;58(6): 346-349.
37. Tayal VS. Tracheostomies. Emerg Med Clin North Am 1994;12 (3):707-727.
38. Law JH, Barnhart K, Rowlett W, de la Rocha O, Lowenberg S. Increased frequency of obstructive airway abnormalities with long-term tracheostomy. Chest 1993;104(1):136-138.
39. Nowak P, Cohn AM, Guidice MA. Airway complications in patients with closed-head injuries. Am J Otolaryngol 1987;8(2):91-96.
40. Woo P, Kelly G, Kirschner P. Airway complications in the head injured. Laryngoscope 1989;99 (7, Part 1):725-731.
41. Kacmarek RM, Pierson DJ. Assessment and management of airway protection, obstruction, and secretion clearance. In: Pierson DJ, Kacmarek RM, editors: Foundations of respiratory care. New York: Churchill Livingstone, 1992:561-570.
42. Nash M. Swallowing problems in the tracheotomized patient. Otolaryngol Clin North Am 1988;21(4):701-709.

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Consideration of the International Standard for Airway Humidification Using Simulated Secretions in an Artificial Airway

Hideki Miyao MD, Katsuyuki Miyasaka MD, Toru Hirokawa MD, and Taro Kawazoe MD

BACKGROUND: The use of humidifiers with a heating wire incorporated into the inspiratory limb of the ventilator circuit has become commonplace in mechanical ventilatory support. The heating wire in the circuit, however, may represent a potential hazard with regard to inspissation of secretions in the upper airway, especially in an artificial airway, due to reduced relative humidity. The current International Standard for airway humidification specifies only absolute humidity (AH) without regard for relative humidity (RH) or temperature. We studied whether either RH or the temperature range *and* AH should be specified in the Standard to maintain adequate RH. METHODS & MATERIALS: Five kinds of gases with various humidities were passed through an endotracheal-tube model, in which a brown water-soluble gel on dry filter paper was used as a substitute for secretions. Because the gel was hydrated by moisture from the delivered gas, the gel melted and a brown stain spread over the dry filter paper to the extent that the gas could hydrate the gel. The stains were measured and the stained area calculated by computer. The extent of each stained area was assessed with respect to RH, AH, and temperature of the gas. RESULTS: Gases satisfying the recommendations of the International Standard (ie, AH > 30 mg H_2O/L), but with low RH, were no more advantageous for hydrating the gel than a gas (AH < 30 mg H_2O/L) saturated with water vapor (ie, RH = 100%). The extent to which the brown water-soluble gel stained an increasing area of filter paper, which we assessed as the ability to provide humidification, was almost directly proportional to RH rather than AH or temperature. CONCLUSION: The current International Standard for humidification of bypassed airways should specify either RH or the temperature range with AH of the delivered gas. [Respir Care 1996; 41(1):43-49]

Introduction

Airway obstruction due to consolidation of airway secretions has been experienced occasionally in our institution since the introduction of humidifiers with heating wires.[1] Such problems had not been encountered before their introduction. We speculated that these problems were caused by a reduction in the relative humidity (RH) of the delivered gas. In fact, the RH of gas passing through inspiratory tubing of a system incorporating a humidifier without a heating wire is higher than the RH of gas passing through tubing with a heating wire.[2] In a previous paper,[1] we showed that a gas with low RH dehydrated a tracheal model to a greater extent than a gas with high RH, despite their having the same AH. This showed the dehydrating effects of gases using a tracheal model. In the present study, we focused on the hydrating effects of gases using a endotracheal-tube model.

The International Organization for Standardization (ISO)

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states "Humidifiers may be used in patients whose upper airway has been bypassed by a tracheostomy or tracheal tube. Under these conditions, 30 mg/L (30g/m^3) is considered to be the minimum amount of water necessary to prevent inspissation of secretions and mucosal damage."[3,4] The American National Standards Institute (ANSI) also states "For devices that bypass the subject's upper airway, a fluid output of 30 mg/L shall fall within the operating range. A heater may be used for this purpose."[5] These two Standards do not specify the RH or the temperature of a gas. AH, RH, and temperature are interdependent, such that setting any two of these three values determines the remaining value. We contend that because a gas, described by only one of these three factors, is able to have various levels of heat energy that may dehydrate secretions, at least two of the values should be specified in the Standards. The content is important because humidification systems are designed by manufacturers in accordance with the Standards.[6] For instance, the machine used in the present study is one of the most recent models and has a dial that allows the RH to be inadvertently adjusted to an unallowable level (< 60%).

Any gas delivered at less than body temperature inevitably causes some loss of water from the airway mucosa until the gas is able to contain 44 mg/L of water vapor (ie, until the inspired gas reaches the saturation point of 44 mg/L at 37° C). Therefore, under dehydrating conditions, it is important to know where the most dehydration occurs. The removal of water mainly from the upper airway may result in the consolidation of secretions at this point in the airway because the total mucosal surface area in the upper airways is smaller than that in the peripheral airways. The purpose of this study was to clarify experimentally whether RH or AH is the dominant factor contributing to moisturization of secretions in the tracheal tube.

Methods

Heated humidifiers* with a heating wire circuit or without a heating wire system were connected to a ventilator in a room with an ambient temperature of 24° C. The humidification chamber was supplied with distilled water by continuous drip without changing the water level or temperature throughout the experiment. An endotracheal-tube model was constructed from a corrugated breathing tube 15 cm in length and 2.2 cm in diameter, in which round filter paper (9-cm diameter) was inserted and put in contact with the inner surface of the tube. As a substitute for secretions, 0.5 mL of water-soluble povidone-iodine ointment gel (Isodine gel*) was put on the dry filter paper in the endotracheal tube. The gel was positioned on the filter paper with a 2.5-mL plastic syringe, taking care to make each surface area constant (5 mm in height and hemispheric in shape) and was positioned at the lowest point in the endotracheal tube in order to eliminate gravitational variables. The endotracheal-tube model was connected to a breathing circuit in which the exhalation valve was occluded (Fig. 1). Five types of pure oxygen gas were passed through the endotracheal tube for 60 minutes at a continuous flow of 30 L/min after measuring the temperature and RH of these gases at the midpoint of the endotracheal-tube model with an electric hygrometer that had been calibrated by the method specified in JIS Z8800 (6-2-2), using saturated salt solutions.[7]

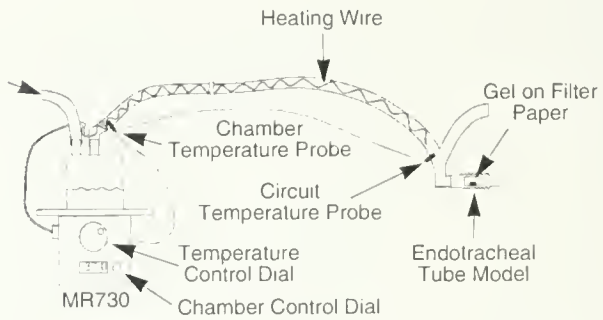

Fig. 1. Experimental set-up. Circuit temperature is set by temperature control dial ranging from 30° C to 39° C. Humidification chamber temperature is set by chamber control dial ranging from −5 to +2. Circuit temperature = 36° C, Chamber control dial = −5 means that humidification chamber temperature is at 31° C and heating wire heats gas to 36° C at circuit temperature probe.

Gas 1 was a dry gas that bypassed the humidifier. Gas 2 passed through a humidifier with a heating wire circuit. The circuit temperature (measured by a temperature probe located about 10 cm from the patient connection port, Fig. 1) was set at 36° C. The chamber control dial was set at −5, meaning that the humidification chamber temperature was at 31° C and the heating wire heats the gas up to 36° C. Gas 3 passed through a humidifier with a heating-wire circuit. The circuit temperature was set at 39° C and the chamber control dial at −5, meaning that the humidification chamber temperature was at 34° C, and the heating wire heated the gas to 39° C. Gas 4 passed through a humidifier with a heating-wire circuit. The circuit temperature was set at 36° C and the chamber control dial at −1, meaning that humidification chamber temperature was at 35° C and the heating wire heated the gas to 36° C. Gas 5 passed through a heated humidifier without a heating-wire circuit. The chamber control dial was set at 8.5, which was merely an arbitrary indicator of humidification-chamber temperature found on this particular humidifier. To prevent the condensate in the circuit from entering the endotracheal-tube model, the circuit was positioned below the model. The temperatures mentioned were the dial settings. Actual temperature and humidity were measured at the midpoint of the model with the electric hygrometer.

* Products and suppliers are identified in the Product Sources section at the end of the text.

After a 1-hour exposure to each gas, the filter paper was removed from the model and the portion of filter paper infiltrated by the water-soluble gel was photographed. The dimensions of the solid gel was subtracted from those of the stain. Areas were calculated by computer using area-calculation software called NIH Image.

Three other trials were carried out to eliminate the effect of temperature and spontaneous melting of the gel itself. First, a dry gas was passed through a humidifier with a heating-wire circuit and humidification chamber with no water. The circuit temperature was set at 39° C, and the chamber control dial at –5. Second, a gel on filter paper (the control) was left alone in the model for 1 hour at an ambient temperature of 24° C. Third, a gel on filter paper was left alone for 24 hours at 24° C.

The AH was calculated based on the formula in Helmholz's work[8] that shows the relation between temperature, water content, and vapor pressure in a saturated gas.

AH = [water content in saturated gas at T (°C)] × RH/100,

where: AH = absolute humidity in mg/L, T (°C) = temperature at the midpoint of the endotracheal-tube model, and RH = relative humidity in %.

Results

Table 1 shows the actual temperature, RH measured at the midpoint of the endotracheal-tube model by the electric hygrometer, the calculated AH, and the area values of stained filter paper for five types of gases. Gas 1, Gas 2, and Gas 5 did not satisfy the recommendations of the International Standard; that is, their AHs were lower than 30 mg/L.

Figures 2, (a), (b), (c), (d), and (e) show the gel on the filter paper after 1 hour of exposure to Gases 1, 2, 3, 4, and 5, respectively. Figure 2(a) shows that the gel absorbed no water. Figure 2(b) shows that the gel coagulated as in Figure 2(a), and there is a small stain around the gel. Figure 2(c) shows some stain around the gel. Figure 2(d) shows the gel melting to a certain degree, with a larger stained area than that of Gas 3. The conditions used to obtain the data in this figure were within the limits recommended by the operation manual of the equipment.[9] Figure 2(e) shows the gel melted. The stained area is the largest among the 5 gases. Although water infiltrated over the brown stained area of Figure 2(e), the value of this stained area (Table 1) does not include this water infiltration. Many droplets of water condensate formed in the circuit of Gas 5 but not in the breathing tube for the four other gases. In other words, Gas 5 was fully saturated with water vapor.

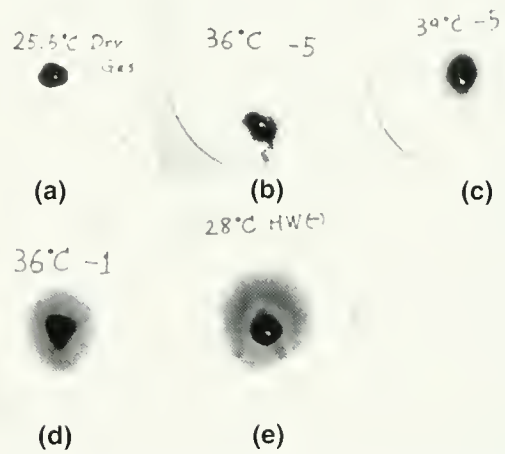

Fig. 2. Ointment gel on dry filter paper after 1-hour exposure to each gas. (a) 25.5° C dry gas (Gas 1). Humidifier not used. (b)-(d): Gases 2-4. 36° C; –5 indicates set-temperature and chamber control dial of MR730. Actual temperature differed from this setting. Details are described in Table 1. (e) Gas 5. 28° C indicates actual temperature at midpoint of endotracheal tube model. HW(–) indicates breathing tube without heating wire. Humidifier used was MR428.

Figure 3 shows that the stained areas of Gas 2, 3, 4, and 5 are almost in direct proportion to RH, but not to AH or temperature.

The stained areas of both the 39° C dry gas and the control were virtually identical to that of Gas 1. The 24-hour ex-

Table 1. Temperature (at Humidifier Chamber, Circuit, and Tracheal Model), Relative Humidity, Calculated Absolute Humidity, and Stained Area for Each Gas.

	Humidifier	Chamber Temperature (°C)	Circuit Temperature (°C)	Tracheal Model Temperature (°C)	Relative Humidity (%)	Absolute Humidity (mg/L)	Stained Area (cm^2)
Gas 1	NA*	NA	NA	25.5	4.5	1.1	0.0
Gas 2	MR730	31.1	36.0	35.5	58	23.6	0.6
Gas 3	MR730	34.3	39.2	38.0	66	30.5	2.0
Gas 4	MR730	35.0	35.9	35.0	80	31.7	8.7
Gas 5	MR428	NA	NA	28.0	100	27.2	17.2

*NA = not applicable.

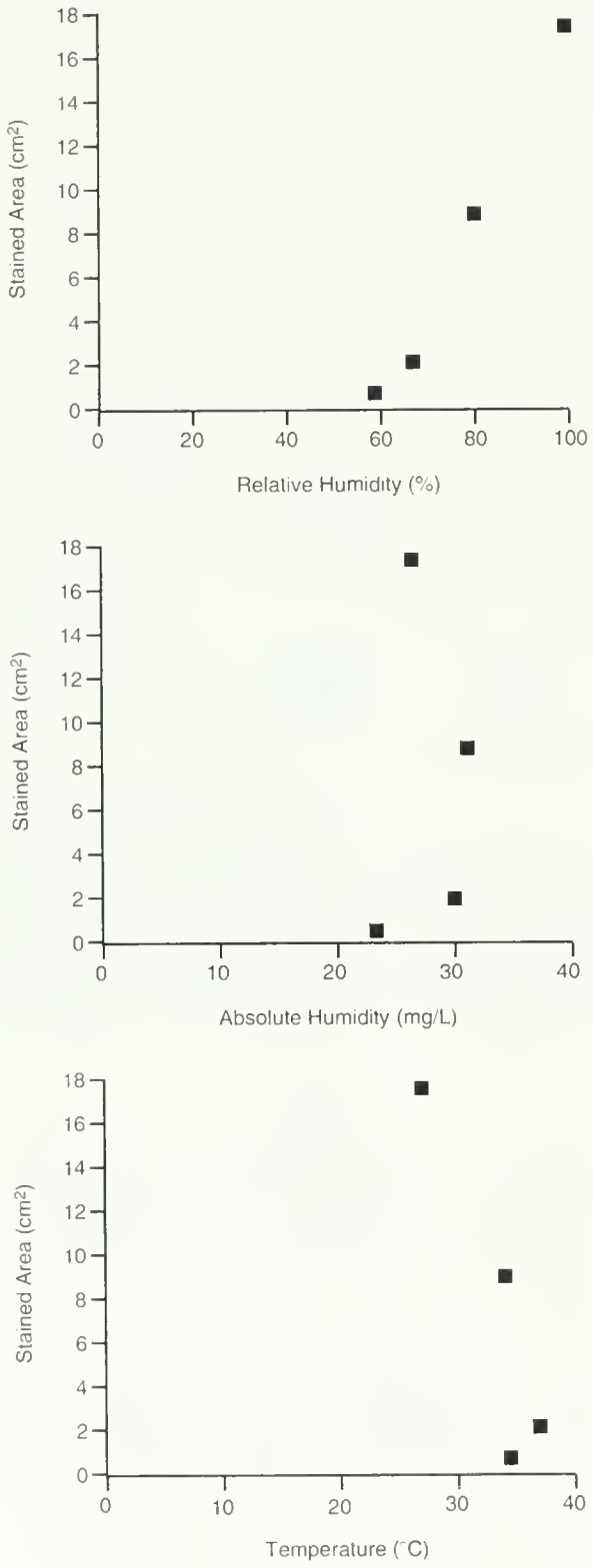

Fig 3. Stained area versus relative humidity, absolute humidity, and temperature. Stained areas are proportional to relative humidity, not to absolute humidity or temperature.

posure of the gel in room air revealed that the stained area was virtually identical to that of Gas 4.

Discussion

It may be helpful to reiterate the relationship between humidity and temperature for the purpose of discussion. Absolute humidity is the amount of moisture present in a volume of gas and is described in terms of mg/L or mg H_2O/L. Relative humidity is the ratio of the amount of moisture in the gas to the maximum amount that the gas can contain at the same temperature, stated as a percentage. As the temperature goes up, AH remains constant, but RH is reduced because the amount of water in the gas remains unchanged, whereas the maximum amount that the gas can contain has increased.

Physiology

In normal nasal respiration, inspired gas in the subglottic region has a temperature of 32-34° C, a RH of 100% and an AH of 36-40 mg/L,[10] or a temperature of 32.3 ± 0.8° C and a RH of 98-99%.[11] It should be noted that the gas is almost completely saturated (98-100% RH). Air is inspired across a wide range of temperatures and humidities, but alveolar gas is fully saturated with water vapor at body temperature. Therefore, a gradient exists between the nose and the point called the isothermic saturation boundary (ISB)[12] where inspired gas reaches 37° C and 100% RH (44 mg/L). This is thought to be just below the carina in adults normally, but the exact point varies with changes in heat and moisture content and in the tidal volume of inspired gas. Above the ISB, the airway acts as a countercurrent heat and moisture exchanging system facilitated by turbulent gas flow.[13] Turbulence that occurs in the upper airway is an important factor in promoting the rapid transfer of heat by convection because air conducts heat poorly, and radiation is a relatively slow means of heat exchange.[14]

Endotracheal intubation bypasses most of the normal heat and moisture exchanging areas, thus eliminating their ability to condition inspired gases. During expiration, gas from lower in the lungs (heated to core temperature and fully saturated with water vapor) gives both heat and water (in the form of condensation) to the mucosa. We readily admit that a 1-way flow of gases, as was used in the model, does not mimic physiologic conditions. However, if such a countercurrent system were completely satisfactory, inspissation of secretions would never occur, even after long-term exposure to dry gas, and, consequently, humidifiers would not be necessary.[6] Inspissation of secretions, therefore, is caused primarily by the characteristics of the inspired gas. This is the reason why we adopted the unidirectional gas flow.

Humidifier

When a humidifier without a heating-wire-circuit system

is used, it is not necessary to set the lower limit of RH because RH will always be high. The reason is that the delivered gas is cooled by ambient air along the inspiratory limb of the circuit, and the maximum amount of water vapor that the gas can contain at that temperature becomes reduced, resulting in condensation of water vapor in the breathing tube. This means 100% RH at the given temperature.

A heating-wire system was developed in an attempt to eliminate the condensate. The advantages of this system are ease of nursing, a possible reduction in the number of nosocomial infections resulting from aspiration of contaminated condensate, and, most importantly, high AH of the delivered gas. However, if the delivered gases are heated higher than the temperature of the humidification chamber, it can make RH low enough to inspissate secretions in the upper airway, especially in a tracheostomy tube or an endotracheal tube, despite the presence of high AH.

The present study shows that the stained areas infiltrated by the water-soluble gel are almost directly proportional to RH, not to AH or temperature. This suggests that RH is the dominant factor with regard to the drying[1] or moisturizing of secretions in the upper airway. Gas 5 (28° C, 100% RH, 27.2 mg/L) would moisturize the secretions at the proximal inlet of the airway despite an AH of 27.2 mg/L, which does not meet the Standards. This gas, however, will in turn deprive the peripheral airway of water because of the low AH. It should be noted that saturated gas (100% RH) has no ability to deprive the airway of water until it is warmed. From the physiologic viewpoint, when this gas exits the tip of the endotracheal tube, heat is rapidly transferred to the gas by turbulent convection. However, RH does not decrease because the turbulent gas also contains moisture from the mucous membranes. Saturated gas will not have a dehydrating effect until the temperature increases. Such a gas would pass through the bronchial tree, removing water gradually as the temperature approaches the isothermal saturation point (37° C, 44 mg/L). In contrast, Gas 3 (38° C, 66% RH, 30.5 mg/L), which meets the standards, already has the potential to dehydrate because it is not saturated. Therefore, this gas deprives more water from the upper airway than does Gas 5. Then, secretions in the upper airway become dry. However, Gas 3 removed less water (44 – 30.5 = 13.5 mg/L) from the lung than Gas 5 (44 – 27.2 = 16.8 mg/L). Nevertheless, Gas 5 had a much stronger ability to hydrate the gel than Gas 3 in the uppermost part of the airway. This is because of the high RH of Gas 5. Figure 4 shows a possible AH curve and mucosal surface area that appears to have received little discussion in the literature, except for one report[1] saying that AH is essential in determining the total amount of water deprived from the patient's lung, and that RH is decisive in determining the location where most of the water is deprived. Although secretions are replenished with water vapor during the expiratory phase, they might become dry if the amount of water removed exceeds the amount replenished. This is mainly because desaturation of the inspired gas can remove more water and partly because the high temperature of the inspired gas might reduce water replenishment during the expiratory phase without cooling the expired gas. Note that the present results must be discussed in relation to the endotracheal tube. Figure 4 shows, however, that secretions in the endotracheal tube could be exposed to a more undesirable environment than those in the trachea because there is no source of water other than the secretions.

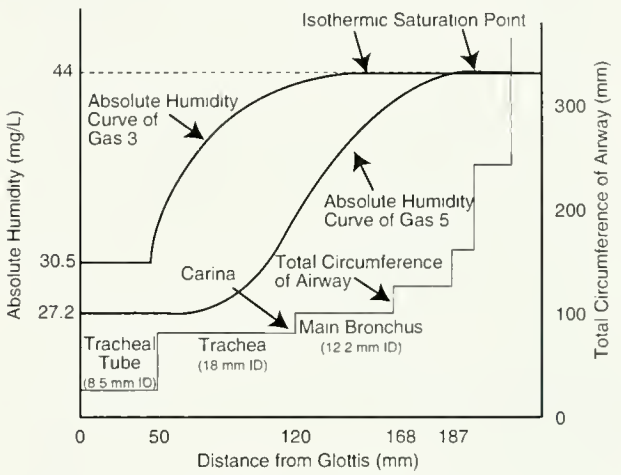

Fig. 4. Possible absolute humidity (AH) curve of Gas 3 and Gas 5 in inspiratory phase and total circumference of airway. Due to reduced relative humidity, Gas 3 (38° C, 66%, 30.5 mg/L) has greater ability to dehydrate mucosa than Gas 5 (28° C, 100%, 27.2 mg/L). Gas 3 would deprive the trachea of the greatest amount of water, resulting in steeper rise in AH in this area. AH increases because water is supplied from tracheal mucosa. Mucosal surface area, which is not proportional to total cross-section of airway but to total circumference, rapidly expands from carina. Steplike line represents total circumference of airway (calculated by multiplying diameter × π × airway generation number) adapted from data in Weibel's work,[22,23] which shows the average diameters and lengths of Human Adult Airway Model "A." Right side of vertical axis refers to total circumference of airway in mm. Horizontal axis refers to distance from glottis in mm. Mucosal surface area is calculated by multiplying circumference by distance; mucosal surface area can be obtained by integrating area below line of total circumference of airway. If speculation about shape of AH curve for each gas is correct, amount of water deprived per unit area of tracheal mucosa by Gas 3 would be larger than that by Gas 5, although total amount of water deprived by Gas 3 (13.5 mg/L = 44 – 30.5) is actually smaller than that deprived by Gas 5 (16.8 mg/L = 44 – 27.2).

Standards

Ingelstedt,[11] Sara and Curie,[15] Chatburn and Primiano,[10] and Chatburn[16] measured the temperature and RH of normal nasal inspirations in the trachea. The measured RHs are all above 96%. ISO and ANSI only recommend that AH be above 30 mg/L. The AARC (American Association for Respiratory Care) recommends that inspired gas be delivered at 33 ± 2° C and provide a minimum of 30 mg/L of water vapor.[17] These

values correspond to 76-94% RH. Forbes[18,19] noted that mucus flow in animals was not affected by humidity when AH was kept above 33 mg/L and/or RH was > 75%. These findings are reflected in the British Standards.[20] These standards or guidelines appear to be based on the findings of the functions of cilia and mucus flow in the presence of various humidity conditions. The ciliated epithelium and mucous membrane in the upper airway have defensive mechanisms against unfavorable circumstances, eg, sweeping particles and coagulated mucus out of the respiratory tract and increasing mucus production and mucus flow. The inner surface of the artificial airway, however, becomes easily obstructed when secretions in the artificial airway are exposed to a dehydrating condition. Chatburn and Primiano,[10] Chatburn,[16] and Tsuda[21] recommended 100% RH for bypassing the upper airway. We also recommend 100% RH, and a small quantity of condensation in the inspiratory limb of the breathing circuit is the only reliable indication of 100% RH under the present circumstances. AH, RH, and temperature are interrelated, and once any two of these three factors are set, the third one is determined; ie, the energy level of the gas is determined by any two of the three variables. A gas described by only one variable, such as a gas with an AH of 30 mg/L, is able to have various energy levels. For instance, a gas with an AH of 30 mg/L at 38° C has higher heat energy than a gas with the same AH at 30° C. It is this heat energy that deprives the artificial airway of water. To clarify the concept of thermal-energy level of a gas, we believe that two of the three variables must be specified in the Standards, as is the case in the AARC Guideline. In contrast, ISO and ANSI Standards state only the method for measuring water content. This may be because measuring the RH of a gas is technically less accurate than measuring the AH. Because temperature can be measured accurately, RH can be maintained at an appropriate level by adding a temperature specification to the Standards.

In order to understand the factors contributing to drying or hydrating of the secretions in the artificial airway, it is first necessary to simplify the experimental model. Although the methodology used in the present study has several limitations, we believe that the ideas contained in this study expose a "blind spot" in daily clinical practice. However, before the results of the present study can be applied to clinical practice, in-vivo studies and further investigations of appropriate temperature and RH are necessary, although it may be difficult to control all the conditions that determine humidity.

Conclusion

Relative humidity is the predominant factor when it comes to humidifying the secretions in an artificial airway. Therefore, we believe that the current International Standard for humidification of bypassed airways should specify either the relative humidity or the temperature range *and* the absolute humidity of the delivered gas.

PRODUCT SOURCES

Humidification System:
Humidifier with heating wire: MR730-S, MR428, and MR310, Fisher & Paykel, Auckland, New Zealand

Ventilator & Circuit:
E150 Ventilator and PBC 300A Circuit, Newport Medical Instruments, Newport Beach CA, USA

Endotracheal-Tube Model Made from Disposable Corrugated Circuit:
001849 Inspiron, Lincolnshire IL, USA

Povidone-Iodine Ointment Gel:
Isodine gel®, Meiji Seika Co, Tokyo, Japan

Electric Hygrometer:
HMP 133Y, Vaisara, Helsinki, Finland

REFERENCES

1. Miyao H, Hirokawa T, Miyasaka K, Kawazoe T. Relative humidity, not absolute humidity, is of great importance when using a humidifier with a heating wire. Crit Care Med 1992;20(5):674-679.
2. Martin C, Papazian L, Perrin G. Performance evaluation of three vaporizing humidifiers and two heat and moisture exchangers in patients with minute ventilation >10 L/min. Chest 1992;102(5): 1347-1350.
3. ISO 8185:1988(E) 60 Liquid output, pp 19, Annex O. Humidifiers for medical use-Safety requirements. International Organization for Standardization, 1988.
4. ISO 8185:1988(E) 60. 1 Liquid output, pp 14, Section 10, Humidifiers for medical use-Safety requirements. International Organization for Standardization, 1988.
5. ANSI Z79.9-1979: American National Standard for Humidifiers and Nebulizers for Medical Use 3.1.1.2, 3.1.1 Fluid Output, 3. New York: Requirements, American National Standards Institute, 1979:8.
6. Miyao H, Miyasaka K. Relative humidity with heating wire (letter). Crit Care Med 1993;21(10):1613-1615.
7. Greenspan L. Humidity fixed points of binary saturated aqueous solutions. J Res Nat Bur Stand 1977;81:86-96.
8. Helmholz HF Jr, Burton GG. Applied humidity and aerosol therapy. In: Burton GG, Hodgkin JE (editors). Respiratory care. Philadelphia: Lippincott Co, 1984:379-394.
9. Using the Chamber Control. MR700, MR720, MR730, 7.3 respiratory humidifiers operating manual, version 2.0. Auckland, New Zealand: Fisher & Paykel Co, 1992:26.
10. Chatburn RL, Primiano FP Jr. A rational basis for humidity therapy (editorial). Respir Care 1987;32(4):249-253.
11. Ingelstedt S. Studies on the conditioning of air in the respiratory tract. Acta Otolaryngol 1955;131(suppl):7-55.
12. Dery R, Pelletier J, Jacques A, Clavet M, Houde JJ. Humidity in anaesthesiology. III: heat and moisture patterns in the respiratory tract during anaesthesia with the semi closed system. Can Anaesth Soc J 1967; 14(4):287-298.
13. Shelly MP, Lloyd GM, Park GR. A review of the mechanisms and methods of humidification of inspired gases. Intensive Care Med 1988;14(1):1-9.
14. Walker JEC, Wells RE Jr, Merrill EW. Heat and water exchange in the respiratory tract. Amer J Med 1961;30:259-267.
15. Sara C, Currie T. Humidification by nebulization. Med J Aust 1965;1: 174-179.
16. Chatburn RL. Physiologic and methodologic issues regarding humidity therapy (editorial). J Pediatr 1989;114(3):416-420.
17. American Association for Respiratory Care. Clinical practice guideline: humidification during mechanical ventilation. Respir Care 1992; 37(8):887-890.

18. Forbes AR. Humidification and mucus flow in the intubated trachea. Br J Anaesth 1973;45(8):874-878.
19. Forbes AR. Temperature, humidity and mucus flow in the intubated trachea. Br J Anaesth 1974;46(1):29-34.
20. British Standards Institution. Specifications for humidifiers for use with breathing machines. Publication No. BS 4494. London: The Institution, 1970.
21. Tsuda T, Noguchi H, Takumi Y, Aochi O. Optimum humidification of air administered to a tracheostomy in dogs. Scanning electron microscopy and surfactant studies. Br J Anaesth 1977;49(10):965-977.
22. Weibel ER. Morphometry of the Human Lung. Heidelberg: Springer-Verlag. 1963:139.
23. Weibel ER. The lung: scientific foundations. New York: Raven Press, 1991: 711-720.

CORRECTION

One of the books included in Op't Holt TB. Comparative review of respiratory care equipment texts. Respir Care 1995;40(8):876-878 was cited incorrectly. The textbook should be

Equipment Theory for Respiratory Care, by Gary C White. Hardbound, 505 pages. Albany: Delmar, 1992. $48.95.

The book was inadvertently listed as published in 1995. The new edition was released December 1, 1995. The comments of the reviewer may not apply to the 1995 edition. We regret the error.

What Have We Learned about Lung Injury from the Management of Near-Drowning?

Jerome H Modell MD

Introduction
Seawater vs Freshwater Aspiration
Drowning & Near-Drowning without Aspiration
Ventilating the Victim of Near-Drowning
Outcome in Near-Drowning
Related Studies
Water Aerosol
Gastric Contents
Perfluorinated Hydrocarbons
In Conclusion

Introduction

Although Karpovich[1] observed the anatomical changes that occurred within the lungs of rodents after aspiration of both fresh water and seawater in 1933, it was not until the classic work of Henry Swann and his co-workers in the 1940s[2-4] that we began to appreciate many of the physiologic changes that occur with the aspiration of fresh water and seawater. In his classic report,[2] Dr Swann reported that oxygen content decreased during drowning, but he gave particular emphasis to the electrolyte changes resulting from the hypovolemia that occurred during seawater aspiration and from the hypervolemia that occurred during freshwater aspiration. Further, he attributed the demise of his experimental animals after seawater aspiration essentially to the heart failure and massive pulmonary edema that result in lack of oxygenation; whereas, he attributed their deaths after freshwater aspiration to hypochloremia, hyponatremia, and hyperkalemia that resulted in ventricular fibrillation.

It was not until the early 1960s that we began to more completely appreciate the pulmonary effects of water aspiration. In 1960, Redding, Voight, and Safar[5] reported on the use of intermittent positive pressure breathing as treatment for experimental drowning. During the next 3 years, Colebatch and Halmagyi[6-8] reported on the decreased compliance, increased resistance, and reflex pulmonary hypertension that occurred after aspiration of liquid by animals. The Wellcome Prize Essay by Fuller in 1962[9] extensively summarized the changes in drowned victims observed on autopsy at the Armed Forces Institute of Pathology. Fuller's work demonstrated that, in drowning victims, aspiration of not only water but also of contaminants such as algae, sand, or even partially digested food particles could occur.[10] He noted the significant pulmonary edema that occurs and the presence of secondary infection. He also observed distended alveoli in some victims and the deposition of hyaline material in short-term survivors.

I treated my first severe drowning victim in 1962,[11] and, beginning in 1963, began to study the effects of aspiration of not only seawater and fresh water, but also other types of fluids. In this paper, I focus predominantly on the experiences that my colleagues and I have had in the research laboratory and in treating patients in the emergency department and critical care unit, since those days in the early '60s.

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Seawater vs Freshwater Aspiration

The report by Swann and others[2] showing the rapid onset of an increase in whole blood density after seawater aspiration and a decrease in whole blood density after freshwater aspiration led the way for a physiologic approach to the aspiration of water during drowning. As a result of Swann's studies, for approximately two decades, the literature emphasized the changes in serum electrolyte concentrations that would result from these rapid shifts of fluid and their effect on cardiac activity, despite the fact that there were no data on humans that supported this position.

In a series of 91 near-drowned victims, I and others reported that the concentrations of serum chloride, sodium, and potassium of these patients when they arrived in the emergency room did not differ significantly among victims of fresh water, seawater, or brackish water aspiration.[12] There was a tendency for the serum sodium and chloride concentrations to be slightly higher after seawater aspiration, but in no case were the levels thought to be life threatening. These observations led us to conduct experiments in the laboratory on anesthetized animals. Known quantities of water were aspirated, and blood was obtained from the arch of the aorta at regular intervals, beginning at 1 minute after aspiration, to follow any changes in serum electrolyte concentrations.[13-15] We found a transient decrease in serum chloride and serum sodium 1 minute after aspiration of distilled water, regardless of whether it was chlorinated at a level of 5 parts per million; however, within 10 minutes the concentrations of these two extracellular electrolytes were no different from those in animals that had aspirated normal saline solution. The serum potassium increased in all three groups, but more so in the freshwater groups 3 to 5 minutes after aspiration, but, again, by 10 minutes after aspiration, had returned to a normal level. The volume of fluid aspirated in these experiments was approximately twice the tidal volume of the experimental subjects.[15]

When we studied experimental animals under conditions of total immersion, we observed significant changes in the concentration of serum electrolytes, both with aspiration of fresh water and seawater; thus, we were able to repeat the earlier observations of Swann and others.[13,14] Our conclusion, at that point, based on comparing our observations in humans with the experimental data, was that humans who had been resuscitated at the scene of the accident and brought to the emergency room more than likely aspirate 22 mL/kg of water or less.[12,16]

Joseph Davis, Medical Examiner for Dade County, and I participated in the autopsies of 118 consecutive drowned victims who had been retrieved from water but could not be revived by resuscitation efforts.[17] The levels of serum chloride and serum sodium in blood from the left side of the heart of these victims, when compared with our experimental values for which the volume of aspirate was known, showed that only 15% of victims who died in the water had serum electrolyte concentrations that deviated by more than 20 mEq/L from the midrange of normal. These findings suggested, therefore, that 85% of persons who died at the scene also aspirated 22 mL/kg of water or less. Thus, we concluded that it is not the changes in serum electrolyte concentrations that lead to the demise of these victims, but rather the lack of oxygenation at a level compatible with life.

Early reports on drowning also speculated that, with freshwater aspiration, there would be enough absorption of fresh water to cause massive hemolysis of red blood cells, which results in an extremely high serum potassium concentration. With the exception of one patient with a plasma hemoglobin concentration of 500 mg%, we did not observe a plasma hemoglobin level greater than 100 mg% in any other patient.[18] We then sought to separate, in an experimental model, the effect of significant hemodilution with fresh water when oxygenation was normal from that of multiple conditions that would have produced concurrent profound hypoxia. We found that, in well-oxygenated animals, a significant increase in plasma hemoglobin concentration did not occur, despite the infusion of 44 mL/kg of distilled water directly into the venous circulation. However, when simultaneous airway obstruction occurred with the intravenous instillation of this amount of water or when water was aspirated into the lung, which compromised oxygenation, then a plasma hemoglobin concentration exceeding 900 mg% was not uncommon.[19]

In the same animals in which persistent electrolyte changes could not be demonstrated beyond 10 minutes after aspiration of 22 mL/kg of fresh water, we did demonstrate that profound hypoxemia occurred within 1 minute of aspiration. When the animals either survived this insult or were resuscitated and thereafter breathed spontaneously, arterial oxygen tension (P_{aO_2}) did not improve over the 60-minute experiment, whether the liquid aspirate was normal saline solution, distilled water, or chlorinated distilled water.[15] A transient increase in arterial carbon dioxide tension (P_{aCO_2}) also occurred within 3 to 5 minutes of aspiration. However, by 10 minutes after aspiration, P_{aCO_2} was approaching normal and, by 30 minutes, did not differ from before aspiration. As might be expected, an immediate combined metabolic and respiratory acidosis occurred, and a metabolic acidosis persisted, which coincided with the period of hypoxemia.[15]

Because seawater is at least 3 times more concentrated in regard to electrolyte concentrations than is blood, our experiments were compatible with those of Swann and others, which showed that fluid moved from the vascular space into the lung itself.[14] Routinely, when experimental animals aspirated seawater and we attempted to drain this fluid by gravity, more liquid was drained than had been experimentally instilled. What we did not appreciate early on was the rapidity with which pulmonary edema occurred due to this significant fluid shift across the alveolar capillary interface. In companion studies, we attempted to look at changes in blood vol-

ume after seawater aspiration by utilizing the radio-iodinated serum albumin (^{131}I) technique. The rapidity with which pulmonary edema occurred after seawater aspiration was astounding: In less than 3 minutes after injection of ^{131}I into the vascular space, significant amounts of the radioactive tracer were detected in suctioned tracheal fluid. The extent of albumin leak was apparent by the false high readings of calculated blood volume due to the loss of tracer from the vascular space.[14]

After freshwater aspiration, the aspirated fluid, for the most part, is absorbed into the circulation with equal rapidity. Within 3 minutes of instillation of fresh water into the trachea of our experimental models, only insignificant amounts of water could be reclaimed by gravity drainage.[13] Despite the fact that the water was absorbed rapidly, profound hypoxemia was present, which suggested that the alveoli were unstable and large numbers had collapsed. Therefore, significant intrapulmonary shunt was produced by a mechanism different from that after seawater aspiration.[13,20]

This led us to obtain lung extracts after large quantities of fresh water, seawater, and normal saline solution had been aspirated, to analyze the extract for surface tension activity. The extract from animals that had aspirated normal saline solution or seawater, as well as tracheal fluid drained from those animals, showed surface tension characteristics compatible with normal pulmonary surfactant. After freshwater aspiration, however, neither the drained tracheal fluid nor the lung extract showed normal characteristics of pulmonary surfactant. In both of those cases, the minimum surface tension achieved when the film was compressed in a Wilhelmy balance was significantly higher than normal.[21] This, then, would explain the alveolar instability after freshwater aspiration because the alveoli themselves would be deficient in normally functioning pulmonary surfactant and, therefore, would be less likely to remain open on exhalation. Likewise, being deficient in normal pulmonary surfactant would make them more prone to subsequently leak pulmonary edema fluid, which is virtually universally present in such lungs.

Also to be considered is the possible role that cerebral hypoxia and resultant neurogenic pulmonary edema may play in the overall picture of the near-drowned victim.[22] Regardless of whether it is seawater or fresh water that is aspirated, there is significant intrapulmonary shunting secondary to ventilation/perfusion mismatch. When animals breathing room air were compared with those breathing oxygen, the intrapulmonary shunt after seawater aspiration was almost entirely absolute, or true, intrapulmonary shunt; whereas, after freshwater aspiration, it was a combination of absolute and relative intrapulmonary shunt.[20] This supports the concept that the intrapulmonary shunt after seawater near-drowning is secondary to fluid-filled but perfused alveoli; whereas, after freshwater aspiration, it is due to unstable alveoli from lack of normal surfactant activity.

Drowning & Near-Drowning without Aspiration

In 1931, Cot[23] observed that 10% of the drowned victims he autopsied did not appear to have aspirated water. It has been surmised since that time that such patients die of respiratory obstruction secondary to breath holding or laryngeal spasm and suffer cardiac arrest before they take a breath while submerged. We simulated such conditions in an anesthetized animal model and demonstrated that, within 1 minute of tracheal obstruction, P_{aO_2} decreased from a normal average of 90 torr to 40 torr. Within 3 minutes, P_{aO_2} had decreased to 10 torr.[24] Resuscitation of such animals after 5 minutes led to an 80% survival rate without any apparent (ie, subjective) residual neurologic damage.

We have reported that a similar number, namely 12% of near-drowned victims that we have treated, near-drowned without aspiration.[12] This conclusion was based on the fact that their P_{aO_2} with spontaneous air breathing on admission to the hospital exceeded 80 torr. This, however, is a subjective observation. We could not say whether these victims aspirated very small quantities of water, after which the physiologic change in lung function would be relatively insignificant, or, before they became submerged, suffered an event such as trauma, syncope, or cardiac arrhythmia. What is important in these 10% to 12% of victims is that, if they are resuscitated promptly with basic cardiopulmonary resuscitation before irreversible cerebral hypoxia occurs, recovery will be dramatic and complete. For those who actually aspirate fluid, a scenario of far more persistent altered pulmonary function occurs.

Ventilating the Victim of Near-Drowning

The earliest patients we treated for near-drowning in 1963 and 1964 were frequently ventilated by hand with a Mapleson D-type non-rebreathing circuit. Also, we kept continuous positive pressure applied at all times to their airways. This significantly improved oxygenation, but the extent could not be quantitated because of the limited technology at the time. We then applied, early in 1964, what, at that time, we described as "back pressure" to the expiratory valve of a volume-limited ventilator (JH Emerson Co, Cambridge MA), which also consistently improved oxygenation. Subsequently, this type of treatment was reported by Ashbaugh and others as continuous positive pressure breathing (CPPB) or controlled mechanical ventilation with positive end-expiratory pressure (PEEP).[25]

To alter the course of events, we studied the effect of aspiration of sublethal quantities of seawater and fresh water in anesthetized animals and initially gave them mechanical breaths if needed to induce spontaneous breathing. A second group was given controlled mechanical ventilation but with no end-expiratory pressure. A third group breathed spontaneously but with 10 cm H_2O of PEEP. (At the time of these experiments, we categorized this as PEEP; in retrospect, with

the more modern terminology, it would have been continuous positive airway pressure [CPAP]). A fourth group was mechanically ventilated, and 10 cm H_2O of PEEP was also applied to these animals. We noticed a significant increase in arterial oxygenation in the animals that had aspirated seawater, when they were treated with CPAP or with controlled mechanical ventilation and PEEP. There was no significant increase in oxygenation during the treatment period in the animals that breathed spontaneously or were mechanically ventilated without PEEP.[26] Thus the importance of controlled mechanical ventilation with PEEP or CPAP was established as potential treatment for patients suffering from seawater near-drowning.

In a comparable group of animals that aspirated fresh water, once again, there was a significant improvement in oxygenation in those animals that were mechanically ventilated and given PEEP. We did not find the same improvement, however, in animals that breathed spontaneously but with PEEP of 10 cm H_2O.[27]

To better understand why we could not obtain the same beneficial effect experimentally with CPAP in freshwater as in seawater aspiration, we looked at each animal individually in a subsequent experiment rather than the mean and standard deviation data.[28] It became quite clear that, in approximately half of the animals, CPAP did, indeed, have a very beneficial effect. In the other half, in which the effect was not lasting, mechanical breaths instituted with CPAP maintained a beneficial increase in oxygenation and decrease in intrapulmonary shunt.[28] These results can be explained in that the alveoli, after freshwater aspiration, would be unstable due to the changes in surface tension activity of pulmonary surfactant. Thus, a higher peak inspiratory pressure was required to open the alveoli before CPAP could keep them open.

Another observation was that, even though we could reduce the intrapulmonary shunt dramatically with the application of continuous mechanical ventilation and PEEP in experimental animals, oxygen delivery remained at a low level compared with that in the control group.[29] This correlated with a decrease in cardiac output that occurred when mechanical ventilation under these circumstances was applied to the airway. Our attempts to increase the cardiac output with intravenous dopamine did not increase oxygen availability. When these animals were given a large infusion of crystalloid to increase cardiac output, however, the oxygen availability tripled.[29] We then repeated some of our earlier studies on blood volume changes after aspiration of fresh water and followed the animals for a longer period of time. We were able to show the same immediate increase in blood volume 3 minutes after aspiration of 11 mL/kg and 22 mL/kg of fresh water, as we had done several years earlier. As might be predicted, however, when we analyzed blood volume, it was actually lower 60 minutes after aspiration than before the aspiration of fresh water. This showed that there is a fluid redistribution resulting in relative hypovolemia.[29]

We also observed during mechanical ventilatory support of near-drowned patients that P_{aO_2} varied considerably within brief periods of time with position changes of the patient and/or changes in peak inspiratory pressure and PEEP. Thus, we confirmed that the ventilation-perfusion ratio in these patients varied at any given time and was both position- and pressure-sensitive. This observation influenced our treatment of patients with severe lung infiltrates regardless of the precipitating event.[30]

Outcome in Near-Drowning

In the late 1960s, we introduced the use of prophylactic antibiotic and corticosteroid therapy for the treatment of near-drowned victims.[18] These drugs were introduced on an empiric basis. Once we had the opportunity to study a large series of such patients and to conduct controlled experiments in the laboratory on the use of corticosteroids, we found that survival rate did not differ between those who received these drugs and those who did not.[12,31] We observed that some patients who received corticosteroids developed late lung abscesses, whereas those who were not treated with corticosteroids did not. This may be due to the fact that, as in steroid-treated animals, they were not able to wall-off infection as well as those that were not so treated. This would be consistent with subsequent studies in our laboratory on the aspiration of partially digested foodstuff.[32]

As a consequence of capitalizing on the studies just described and concentrating our therapy primarily on aggressive pulmonary support and monitoring of cardiac performance and fluid balance, we have been successful over the past third of a century in returning victims of near-drowning to active, productive lives, provided irreversible cerebral hypoxia had not occurred before arrival at the hospital. Although we have stopped gathering data on these patients over the past few years, when we published our observations in 1980, we had treated 121 patients and we had lost only 4 patients from pulmonary causes.[33] One of these was in 1964 and another in 1965, both of whom, in retrospect, might have survived had their accidents occurred a few years later when we had a better understanding of the disease and were better prepared to treat it. We also had the opportunity to perform pulmonary function studies on several of our patients after they recovered from near-drowning. We could not demonstrate any consistent, persistent abnormalities.[34]

Related Studies

Water Aerosol

My interest in drowning and near-drowning has led to many related studies of liquid in the airway that I would like to share with you. In the 1960s, humidification of respiratory gases was a problem, and patients who were being ventilated me-

chanically frequently had thick tenacious secretions. With the introduction of heated humidifiers, conditions markedly improved. Further, at about that time, the ultrasonic nebulizer capable of nebulizing a relatively large volume of water into small particle size was introduced. Some physicians incorporated the output from an ultrasonic nebulizer into the ventilatory circuit to try to increase humidification of respiratory gas. We were consulted in the case of one such infant. Despite the fact that his kidneys were putting out water at a maximum rate for his size and age, he continued to gain weight rapidly, had increased infiltrate on chest roentgenogram and a decreasing P_{aO_2}, and appeared to be in congestive heart failure. His laboratory values were consistent with those in experimental animals that had aspirated fresh water in excess of 22 mL/kg.[35] We then applied a similar breathing circuit incorporating the ultrasonic humidifier into an animal model and used fluorescein dye for a marker. What we found was that not only the conducting airway but also sections of the peripheral lung were thoroughly stained with the dye in a brief time, which indicated that these structures were essentially flooded with the aerosol.[36,37] The ultrasonic nebulizer was immediately removed from the ventilator circuit. The infant cleared the excess water through his kidneys and recovered from this episode. Thus, our drowning studies had made a significant impact by demonstrating that humidification of respiratory gas during mechanical ventilation can be overdone.

Gastric Contents

It has been estimated that almost 50% of drowning victims vomit during drowning or resuscitation. The number of such victims who actually aspirate stomach contents is unknown, but clearly this complicates resuscitation. The superimposition of aspiration of stomach contents on the pulmonary injury caused by near-drowning may complicate and prolong respiratory insufficiency. For this reason, we elected to study the effects of aspiration of acid and foodstuff in animal models similar to those we had used for our drowning studies.

The first well-documented description of aspiration of stomach contents was that by Mendelson,[38] who reported the aspiration of stomach contents in women in labor and at the time of delivery. Roberts and Adamsons, studying primates, stated that to cause adverse consequences from aspiration, stomach contents must have a pH $\leq$ 2.5 and be $\geq$ 25 mL in volume (Roberts RB, Adamsons K Jr. Unpublished data). Based on that study, some have suggested that aspiration of liquid by an adult is innocuous when pH is > 2.5 or volume is < 25 mL.[39] Rather than accept that suggestion on face value, we elected to do a mortality study in rodents. Various volumes of liquids with various pHs were injected directly into the trachea while the animals were anesthetized. What we found was that aspiration of liquid, regardless of the pH and the volume, was not innocuous; as one might expect, the lower the pH the lower the volume necessary to produce death.[40]

We also found that, with an aspirate of pH 2.5, death was the exception rather than the rule, even with as much as 6 mL of aspirate per kg body weight (that is, by Roberts and Adamsons' system, about 420 mL in adult humans). Using their volume (which calculates to slightly < 0.4 mL/kg), we observed no deaths when the pH was > 1.4. At a pH of 1.4, approximately one third of our animals died, and with a pH of 1.0 at this volume, 90% died. At the other end of the spectrum, we observed that animals who aspirated liquid with a pH of 3.5 and 5.0 also died provided the volume was great enough.[40] When liquid with an extremely low pH, for example, pH 1.0, was aspirated, it produced a chemical burn in the lung which, in addition to affecting alveoli, destroyed ciliary activity in the trachea.[41] Thus, it would be far more difficult to spontaneously eliminate secretions from the lung. Because the pH of liquid in the stomach is usually greater than 2.5,[42] it is unlikely that "pulmonary burn" per se is often a major problem with aspiration of stomach contents.[42] More likely, it is the volume of fluid and that it may contain foodstuff varying in quantity and in size that produces obstruction and/or foreign-body reactions.

Because of the fear of aspirating highly acidic liquid by patients who are thought to be at risk for pulmonary aspiration of stomach contents, particularly while anesthetizing such patients, it is frequently recommended that their gastric contents be alkalinized.[42,43] Originally administration of particulate antacid was recommended for this purpose; however, studies by Gibbs and others showed that particulate antacid itself, if aspirated, can produce a very significant pulmonary lesion.[41] Therefore, current recommendations are that antacid in nonparticulate liquid form be administered instead.[42,44]

In the early 1960s, Bannister and others suggested that corticosteroid therapy might be beneficial for patients who had aspirated stomach contents because this therapy was thought to decrease the inflammatory response to stomach contents.[45,46] In subsequent studies by us with anesthetized animals, we were not able to demonstrate any significant difference in blood gas exchange after aspiration of hydrochloric acid between animals that were or were not treated with steroid, be it high dose or low dose.[47,48] We did find, however, as in our drowning studies, that mechanical ventilation with PEEP produced a significant increase in arterial oxygenation and decrease in intrapulmonary shunt.[49]

Subsequent studies were done by Wynne and others[32] in our laboratory with an animal model of aspiration of foodstuff partially digested but not of an extremely low pH. They showed that the inflammatory response, as measured indirectly, was less at 1, 3, and 7 days after aspiration in animals that had received a corticosteroid than in their counterparts that were not so treated. However, 21 days later, while the lungs of animals who were not treated with corticosteroids appeared to be normal microscopically, those that were treated had several large areas of granuloma formation. This, we think, is consistent with our observations of human near-drowned victims:

Some of those who had been treated with prophylactic steroid therapy had actually suffered lung abscesses, whereas those not so treated did not.

It is thought by many that aspiration of stomach contents results in a persistent lesion. We have treated several patients thought to have aspirated stomach contents during anesthesia, by applying CPAP aggressively. The duration of pulmonary dysfunction in some was as brief as 24 hours or less.

Perfluorinated Hydrocarbons

Finally, I would like to share with you some experiments we performed 20 to 25 years ago. At that time, we were frequently faced with treating patients who had thick, tenacious secretions; required mechanical ventilatory support; and could not always be treated effectively with bronchoscopy. After publication of the classic, simple experiments of Clark and Gollan[50] demonstrating survival of rodents submerged in oxygenated perfluorinated hydrocarbon liquid and subsequently removed, I had the good fortune of meeting Gollan and having him as a mentor for the next several years. During that time, we collected data on gas exchange during ventilation with oxygenated perfluorinated hydrocarbon liquids.

We built a simple gravity-flow ventilator out of glassware and tubing that was available in our laboratory. After demonstrating that perfluorinated hydrocarbon liquid (FX80, 3M Company) neither altered surface tension characteristics of pulmonary surfactant nor washed surfactant out of animals' lungs ventilated with the liquid,[51] we then proceeded to ventilate anesthetized animals for 30 minutes with FX80 and then to restore them to breathing gas. What we observed was that excellent oxygenation could be maintained, first in animals ventilated with FX80 for 30 minutes and then in a second set of animals whose lungs were ventilated with FX80 continuously for 8 hours after which breathing of gas was restored.[52] The animals did develop respiratory acidosis, inasmuch as we could not adequately remove carbon dioxide with our apparatus.[52] We found, however, that with the use of THAM-E, we could buffer pH change so that the animals could tolerate liquid ventilation.

During liquid ventilation, roentgenograms of the animals' chests showed them to be completely opaque, yet 3 hours after restoration of breathing gas, roentgenograms were normal.[52] In following these FX80-ventilated animals over a longer term, 4 to 10 days, they had lower than normal, but acceptable, P_{aO_2} and a moderate increase in P_{aCO_2}. Roentgenograms during this period had a slight suggestion of more radiolucence than normal, which, in turn, suggested that there might be some problem with air trapping or increase in dead space.[53]

To find whether we might use this technique for washing thick tenacious secretions out of lungs, particularly in patients such as those with severe pseudomonas pneumonia or cystic fibrosis, we injected small pockets of FX80 into mice as an indicator of toxicity.[54] Much to our surprise, during a transient air conditioning failure, we noticed that the mice developed large pockets of gas under their skin. This we attributed to the vaporization of the FX80 into the gaseous stage and presumed that, if that occurred subcutaneously in mice at body temperature, the same may be occurring in the lungs, which thereby produced the blood gas results that we found for the first week or two after FX80 ventilation.

We then approached Allied Chemical Company and asked that they prepare a perfluorinated hydrocarbon liquid for us that had a vapor pressure lower than that of FX80. We repeated our experiments with this new liquid called P1D (Allied Chemical Corporation) and, indeed, we found that, after 1 hour of ventilation with P1D and then restoration to breathing gas, P_{aO_2} was significantly greater after ventilation with P1D than after FX80.[55] When animals breathed 100% oxygen, the difference in effect between these two groups of animals, although still present, could be masked somewhat, which led us to think that there was a greater degree of relative intrapulmonary shunting after ventilation with FX80 than after P1D.

We then conducted an experiment that actually washed thick tenacious liquid material out of the lungs of anesthetized animals, with a marked improvement in arterial oxygenation after reconversion to breathing gas.[56]

Allied Chemical Company then produced another perfluorinated hydrocarbon liquid for us called Caroxin F, which had a boiling point and vapor pressure between those of FX80 and P1D (now renamed Caroxin D).[57] With Caroxin F, after liquid ventilation followed by breathing gas, animals had no apparent adverse clinical signs immediately after liquid ventilation or later: Between 24 and 168 hours after liquid ventilation, P_{aO_2} exceeded 80 torr while the animals breathed room air.[58] Extensive pulmonary function studies were then conducted on animals who had breathed Caroxin F, at intermittent periods from 1 to 160 days after liquid ventilation. The day after liquid ventilation, despite a decrease in compliance, in total lung capacity, and in single-breath diffusing capacity and an increase in resistance, the average P_{aO_2} was 84 ± 11 torr. No abnormalities were noted in any pulmonary function tests between 3 and 168 days after liquid ventilation, except for a moderate, but clinically insignificant, decrease in single-breath diffusing capacity at 3 and 7 days after liquid ventilation.[58] We followed these animals for a period of 18 months, and, although there was a progressive decrease in the amounts of retained Caroxin F, very small amounts were retained in lung tissue without any apparent interference in lung function.[59] This led us to do extensive long-term studies of electrolyte concentrations; blood chemistries, including those indicating liver and renal function; and liver enzymes. None demonstrated any abnormality.[59,60] Finally, we confirmed that liquid ventilation might be applicable to humans, by ventilating primates with Caroxin D on two separate occasions with successful restoration of breathing air.[61]

We were about to begin studies in humans when new reg-

ulations for the Food and Drug Administration were being promulgated and the Bureau of Devices was being created. After consultation with officials of the new Bureau of Devices, we did not think that it was economically feasible to invest the time and effort necessary to complete the process by which they would authorize experimental use of this technique in humans. Therefore, the project was dropped. We are encouraged now to read new reports on liquid ventilation to improve gas exchange in animal models of disease. We hope our early work will prove of value to this new generation of investigators.[62]

In Conclusion

In conclusion, I would like to express my gratitude to the Program Committee and the membership of the American Association for Respiratory Care for giving me the honor of presenting the 1995 Donald F Egan Scientific Lecture and permitting me to reminisce and share with you our past observations.

REFERENCES

1. Karpovich PV. Water in the lungs of drowned animals. Arch Pathol 1933;15:828-833.
2. Swann HG, Brucer M, Moore C, Vezien BL. Fresh water and seawater drowning: a study of the terminal cardiac and biochemical events. Tex Rep Biol Med 1947;5:423-437.
3. Swann HG, Brucer M. Cardiorespiratory and biochemical events during rapid anoxic death. VI Fresh water and seawater drowning. Tex Rep Biol Med 1949;7:604-618.
4. Swann HG, Spafford NR. Body salt and water changes during fresh and seawater drowning. Texas Rep Biol Med 1951;9:356-382.
5. Redding JS, Voight GC, Safar P. Drowning treated with intermittent positive pressure breathing. J Appl Physiol 1960;15:849-854.
6. Colebatch HJH, Halmagyi DFJ. Lung mechanics and resuscitation after fluid aspiration. J Appl Physiol 1961;16:684-696.
7. Colebatch HJH, Halmagyi DFJ. Reflex airway reaction to fluid aspiration. J Appl Physiol 1962;17:787-794
8. Colebatch HJH, Halmagyi DFJ. Reflex pulmonary hypertension of freshwater aspiration. J Appl Physiol 1963;18:179-185.
9. Fuller RH. The 1962 Wellcome prize essay. Drowning and the postimmersion syndrome. A clinicopathologic study. Milit Med 1963; 128:22-36.
10. Fuller RH. The clinical pathology of human near-drowning. Proc Roy Soc Med 1963;56:33-38
11. Modell JH. Resuscitation after aspiration of chlorinated fresh water. JAMA 1963;185:651-655.
12. Modell JH, Graves SA, Ketover A. Clinical course of 91 consecutive near-drowning victims. Chest 1976;70(2):231-238.
13. Modell JH, Moya F. Effects of volume of aspirated fluid during chlorinated freshwater drowning. Anesthesiology 1966;27(5): 662-672.
14. Modell JH, Moya F, Newby EJ, Ruiz BC, Showers AV. The effects of fluid volume in seawater drowning. Ann Intern Med 1967;67(1): 68-80.
15. Modell JH, Gauh M, Moya F, Vestal B, Swarz H. Physiologic effects of near drowning with chlorinated fresh water, distilled water, and isotonic saline. Anesthesiology 1966;27(1):33-41
16. Modell JH, Weibley TC, Ruiz BC, Newby EJ. Serum electrolyte concentrations after freshwater aspiration. A comparison of species. Anesthesiology 1969;30(4):421-425.
17. Modell JH, Davis JH. Electrolyte changes in human drowning victims. Anesthesiology 1969;30(4):414-420.
18. Modell JH, Davis JH, Giammona ST, Moya F, Mann JB. Blood gas and electrolyte changes in human near-drowning victims. JAMA 1968;203:337-343.
19. Modell JH, Kuck EJ, Ruiz BC, Heinitsh H. Effect of intravenous vs aspirated distilled water on serum electrolytes and blood gas tensions. J Appl Physiol 1972;32(5):579-584.
20. Modell JH, Moya F, Williams HD, Weibley TC. Changes in blood gases and A-aDO_2 during near-drowning. Anesthesiology 1968;29(3): 456-465.
21. Giammona ST, Modell JH. Drowning by total immersion. Effects on pulmonary surfactant of distilled water, isotonic saline, and seawater. Am J Dis Child 1967;114(6):612-616.
22. Layon AJ, Modell JH. Drowning and near-drowning. In: Lundgren C, Miller JN, editors. The lung at depth. New York: Marcel Dekker. In press.
23. Cot C. Les asphyxies accidentelles (submersion, electrocution, intoxication oxycarbonique). Étude, clinique, thérapeutique et préventive. Paris: Éditions Médicales N Maloine, 1931.
24. Modell JH. The pathophysiology and treatment of drowning and near-drowning. Springfield, Illinois: Charles C Thomas, 1971.
25. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE. Acute respiratory distress in adults. Lancet 1967;2(7511):319-323.
26. Modell JH, Calderwood HW, Ruiz BC, Downs JB, Chapman R Jr. Effects of ventilatory patterns on arterial oxygenation after near-drowning in seawater. Anesthesiology 1974;40(4):376-384.
27. Ruiz BC, Calderwood HW, Modell JH, Brogdon JE. Effect of ventilatory patterns on arterial oxygenation after near-drowning with fresh water: a comparative study in dogs. Anesth Analg 1973;52(4): 570-576.
28. Bergquist RE, Vogelhut MM, Modell JH, Sloan SJ, Ruiz BC. Comparison of ventilatory patterns in the treatment of freshwater near-drowning in dogs. Anesthesiology 1980;52(2):142-148.
29. Tabeling BB, Modell JH. Fluid administration increases oxygen delivery during continuous positive pressure ventilation after freshwater near-drowning. Crit Care Med 1983;11(9):693-696.
30. Modell JH. Ventilation-perfusion changes during mechanical ventilation. Dis Chest 1969;55(6):447-451.
31. Calderwood HW, Modell JH, Ruiz BC. The ineffectiveness of steroid therapy for treatment of freshwater near-drowning. Anesthesiology 1975;43(6):642-650.
32. Wynne JW, Reynolds JC, Hood CI, Auerbach D, Ondrasick J. Steroid therapy for pneumonitis induced in rabbits by aspiration of foodstuff. Anesthesiology 1979;51(1):11-19.
33. Modell JH, Graves SA, Kuck EJ. Near-drowning: correlation of level of consciousness and survival. Can Anaesth Soc J 1980;27(3): 211-215.
34. Butt MP, Jalowayski A, Modell JH, Giammona ST. Pulmonary function after resuscitation from near-drowning. Anesthesiology 1970;32 (3):275-277.
35. Tamer MA, Modell JH, Rieffel CN. Hyponatremia secondary to ultrasonic aerosol therapy in the newborn. J Pediatr 1970;77(6): 1051-1054.
36. Modell JH, Giammona ST, Davis JH. The effect of chronic exposure to ultrasonic aerosols on the lung. Anesthesiology 1967;28(4): 680-688.
37. Calderwood HW, Klein EF Jr, Modell JH, Teaque PO, Downs JB. Distribution of nebulized aerosols in spontaneously breathing puppies. Anesthesiology 1974;41(4):368374.
38. Mendelson CL. The aspiration of stomach contents into the lungs and during obstetric anesthesia. Am J Obstet Gynecol 1946;52: 191-205.
39. Roberts RB, Shirley MA. Reducing the risk of acid aspiration during cesarean section. Anesth Analg 1974;53(6):859-868.

40. James CF, Modell JH, Gibbs CP, Kuck EJ, Ruiz BC. Pulmonary aspiration—effects of volume and pH in the rat. Anesth Analg 1984;63 (7):665-668.
41. Gibbs CP, Schwartz DJ, Wynne JW, Hood CI, Kuck EJ. Antacid pulmonary aspiration in the dog. Anesthesiology 1979;51(5):380-385.
42. Gibbs CP, Modell JH. Pulmonary aspiration of gastric contents: pathophysiology, prevention, and management. In: Miller RD, editor. Anesthesia, 4th ed. New York: Churchill Livingstone, 1994: 1437-1464.
43. Wynne JW, Modell JH. Respiratory aspiration of stomach contents. Ann Intern Med 1977;87(4):466-474
44. Gibbs CP, Spohr L, Schmidt D. The effectiveness of sodium citrate as an antacid. Anesthesiology 1982;57(1):44-46.
45. Bannister WK, Sattilaro AJ. Vomiting and aspiration during anesthesia. Anesthesiology 1962;23:251-264.
46. Bannister WK, Sattilaro AJ, Otis RD. Therapeutic aspects of aspiration pneumonitis in experimental animals. Anesthesiology 1961; 22:440-443.
47. Downs JB, Chapman RL Jr, Modell JH, Hood CI. An evaluation of steroid therapy in aspiration pneumonitis. Anesthesiology 1974;40(2): 129-135.
48. Chapman RL Jr, Downs JB, Modell JH, Hood CI. The ineffectiveness of steroid therapy in treating aspiration of hydrochloric acid. Arch Surg 1974;108(6):858-861.
49. Chapman RL Jr, Modell JH, Ruiz BC, Calderwood HW, Hood CI, Graves SA. Effect of continuous positive-pressure ventilation and steroids on aspiration of hydrochloric acid (pH 1.8) in dogs. Anesth Analg 1974;53(4):556-562.
50. Clark LC Jr, Gollan F. Survival of mammals breathing organic liquids equilibrated with oxygen at atmospheric pressure. Science 1966;152(730):1755-1756.
51. Modell JH, Gollan F, Giammona ST, Parker D. Effect of fluorocarbon liquid on surface tension properties of pulmonary surfactant. Dis Chest 1970;57:263-265.
52. Modell JH, Newby EJ, Ruiz BC. Long-term survival of dogs after breathing oxygenated fluorocarbon liquid. Fed Proc 1970;29(5): 1731-1736.
53. Modell JH, Hood CI, Kuck EJ, Ruiz BC. Oxygenation by ventilation with fluorocarbon liquid (FX80). Anesthesiology 1971;34(4): 312-320.
54. Modell JG, Tham MK, Modell JH, Calderwood HW, Ruiz BC Distribution and retention of fluorocarbon in mice and dogs after injection or liquid ventilation. Toxicol Appl Pharmacol 1973;26(1):86-92.
55. Tuazon JG, Modell JH, Hood CI, Swenson EW. Pulmonary function after ventilation with fluorocarbon liquid (Caroxin-D). Anesthesiology 1973;38(2):134-140.
56. Calderwood HW, Modell JH, Ruiz BC, Brogdon JE, Hood CI. Pulmonary lavage with liquid fluorocarbon in a model of pulmonary edema. Anesthesiology 1973;38(2):141-144.
57. Tham MK, Walker RD, Modell JH. Physical properties and gas solubilities in selected fluorinated ethers. J Chem Eng Data 1973;18: 385-386.
58. Saga S, Modell JH, Calderwood HW, Lucas AJ, Tham MK, Swenson EW. Pulmonary function after ventilation with fluorocarbon liquid P-12F (Caroxin-F). J Appl Physiol 1973;34(2):160-164
59. Calderwood HW, Ruiz BC, Tham MK, Modell JH, Saga SA, Hood CI. Residual levels and biochemical changes after ventilation with perfluorinated liquid. J Appl Physiol 1975;39(4):603-607.
60. Calderwood HW, Modell JH, Rogow L, Tham MK, Hood CI. Morphologic and biochemical changes in dogs after ventilation with Caroxin-D fluorocarbon. Anesthesiology 1973;39(5):488-495.
61. Modell JH, Calderwood HW, Ruiz BC, Tham MK, Hood CI. Liquid ventilation of primates. Chest 1976;69(1):79-81.
62. Wilcox DT, Glick PL, Karamanoukian HL, Leach C, Morin FC, Fuhrman BP. Perfluorocarbon-associated gas exchange improves pulmonary mechanics, oxygenation, and ventilation and allows nitric oxide delivery in the hypoplastic lung congenital diaphragmatic hernia lamb model. Crit Care Med 1995;23:1858-1863.

Books, Films, Tapes, & Software

Listing and Reviews of Books and Other Media
Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Stedman's Plus Spell Checker* for Microsoft Word® and Ami Pro®, from Williams & Wilkins, 1995, $99; **Stedman's Electronic Medical Dictionary** for the Macintosh, from Williams & Wilkins, 1994, $129.

> I have a spelling checker,
> It came with my PC;
> It plainly marks four my revue
> Mistakes I cannot sea.
> I've run this poem threw it,
> I'm sure your please too no,
> Its letter perfect in it's weigh,
> My checker tolled me sew.
> —Author Unknown

To an assistant editor who has a background in public relations writing and editing, checking microbiologic, pharmacologic, and general respiratory care spellings, abbreviations, and definitions can be challenging and time consuming—particularly when one only has a hardbound copy of *Stedman's Medical Dictionary* and a run-of-the-mill electronic spelling checker.

But checking medical terminology has become faster and easier now that my computer is equipped with an electronic medical spelling checker and a medical dictionary—**Stedman's Plus Spell Checker** for Microsoft Word® and Ami Pro® and **Stedman's Electronic Medical Dictionary** for the Macintosh.

Each software package is a snap to install. (If I can do it without calling the company's help desk—anyone can.) For the spelling checker, a brief, easy-to-read pamphlet provides directions for installation. A small booklet with step-by-step instructions aids users in the installation of the medical dictionary. Each program took < 15 minutes to completely install.

The **Stedman's Plus** spelling checker's numerous medical and pharmacologic terms are merged with the words listed in the current spelling checker of either Microsoft Word® or Ami Pro®. The program also adds eponymic and statistical terms as well as generic drug names. These features have been helpful for spelling checks of the "Abstracts from Other Journals" column, papers that have been accepted for publication, and, in this month's issue, Cordero et al[1]—a paper with several eponymic terms. Further, I tested the capabilities of the spelling checker for disease names by rechecking a copy of Medin and Ognibene's[2] paper.

Although no author exhibited a particular problem with spelling, I was able to determine the words and abbreviations that were omitted. Although many respiratory care terms are included in the spelling checker, I was disappointed to discover that some important respiratory care abbreviations were highlighted as misspelled words (Table 1). The spelling checker does include many genus and species names. The program offers add and delete keys for updating the spelling checker as needed, thus personalizing the product for each user.

The **Stedman's Electronic Medical Dictionary** has also proved helpful. From any application via the use of menus or 'hot-key' combinations, almost instant access is available to the dictionary. The only catch is that the dictionary has to remain open while one uses an application. The potential hold back for users here is the availability of RAM (1.5 MB is required) and disk space (6.2 MB free space is needed).

From Microsoft Word®, I have been able to highlight a word, depress the appropriate hot-key combination, and immediately find a definition. This has been helpful with words such as "tracheotomy" and "tracheostomy" and other easily confused medical words. I have also found that I can leave **Stedman's Plus** spelling checker with a highlighted word and go quickly to the dictionary to find out whether the word is used correctly.

I was surprised to discover that many chemical abbreviations were omitted from the dictionary and were only used to define other terms. For example, H_2O, P_{aCO_2}, HCl, O_2, CO_2 and others. In order to find these terms in the dictionary, one needs to know the correct, spelled-out form. Table 1 also lists respiratory care abbreviations not included in the dictionary.

As good as these programs are, absolutely nothing can take the place of a watchful eye and skillful, if not artful, use of the English language. As the introductory paragraph of this review illustrates, even correctly spelled words can convey nonsense.

Both software packages may be useful to researchers and clinical practitioners. Other professionals such as medical transcriptionists and secretaries, hospital administrators and public relations officials, students and others may find good use for these economical and easy-to-use software programs.

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REFERENCES

1. Cordero L, Davis K, Warner D, Ayers LW. Ureaplasma urealyticum tracheal colonization and respiratory disease in newborns. Respir Care 1996;41(1):22-29.
2. Medin DL, Ognibene FP. Pulmonary disease in AIDS: implications for respiratory care practitioners. Respir Care 1995;40(8): 832-854

Table 1. Comparison of Respiratory Care Abbreviations Included or Omitted in the *Stedman's Plus* or *Stedman's Electronic Medical Dictionary*.

Terms	Spelling Checker	Dictionary
BiPAP	no	no
cm H_2O or cmH_2O	no	no
ECMO	yes	no
FEV_1	yes	no
P_{aCO}	no	only as part of a definition
S_{aO}	no	no
V_{O_2}	no	yes

[a] Some words are not found in the spelling checker or dictionary because of a difference in font or type style.

*Both are also available for Windows® and IBM compatible computers, from Williams & Wilkins, 351 Camden Street, Baltimore MD 21201-2436, (800) 638-0672

RETROSPECTROSCOPE

The Clouded Crystal Ball

In the preceding Retrospectroscope (1), I suggested one pretty dependable way for a scientist to hold back progress—to be highly respected, authoritative, articulate, internationally famous, and, in his *current* views, hypothesis, prediction, or suggestion, *to be wrong*. I gave a few classic "for instances" of scientists who achieved this combination and the consequences of their "achievement." I'm sure I shocked some readers by suggesting that even the best of scientists or clinicians can, once in a while, be wrong. Everyone knows that statesmen, presidents, kings, generals, admirals, economists, critics of music, art, literature, and theater, sociologists, judges (even of the Supreme Court), FBI, and CIA occasionally make mistakes whose consequences range from modest to catastrophic. But over the past few years, public opinion polls have consistently placed research scientists way at the top of the list of professions rated on their integrity and the public's respect, trust, and esteem for them.

I surely don't want to reduce the public's confidence in scientists to a lower level, such as that accorded to Supreme Court Justices or Congressmen or Presidents, but it *is* important to scientists, to the public, and to their Congressmen to learn whether even eminent scientists may occasionally be wrong—not deliberately or maliciously—but in their estimate or conviction of where the "big pay-off" will be in science.

In the good old days, it didn't affect other scientists (and their work) very much if one scientist tried to be clairvoyant but wasn't, because each scientist was free to ignore or follow up the prediction or advice of another. Times have changed, and with change has come greater probability that predictions on where to put the public's money will do good or harm or both. When Congress set up Institute after Institute in the NIH (presumably with advice from scientists), it changed priorities for research for any scientist who needed NIH support for his work. When, again with advice from scientists, Congress greatly increased the budgets of one or two Institutes and decreased the research buying power of other Institutes, it again re-ordered national priorities. Whenever Congress earmarked any part of its appropriation for a specific project, it again reordered priorities. When one of the Institutes assigned more of its money for contract-supported or commission-directed or panel-recommended research, it again reordered the pattern of biomedical research.

For good, for bad, for both? No one knows. For a couple-billion-dollar organization operated by scientist-administrators, with the advice of working scientists, and for the over-all advance of science, it is remarkable that the NIH does little or no continuing research on the effectiveness of new ways of setting priorities in achieving its goals. The data on clairvoyance in science are all in their files—from the day of the first NIH research contract and the first commissioned-mandated research program to today. How much money was awarded to each? for what? on what time schedule? on or against whose advice? How many have or have not made it to the finish line? What exciting new ideas, concepts, diagnostic technics, and cures have come from these new ways of supporting research? How many have made it to Stockholm and the Nobel Prize? Is the huge cancer research program on schedule?

Until someone objectively analyzes recent performances in the art of clairvoyance, it seems worthwhile to look into the Retrospectroscope and see whether eminent scientists have ever been wrong in their predictions of what's going to be terribly important or utterly worthless.

Some Predictions In Biomedical Science

On Poliomyelitis Vaccine

Simon Flexner, Director of the Rockefeller Institute for Medical Research, was quoted in an article in the *New York Times* in 1911, which said (2):

> The Rockefeller Institute ... believes that its search for a cure for infantile paralysis is about to be rewarded. Within six months, according to Dr. Simon Flexner, definite announcement of a specific remedy may be expected. "We have already discovered how to prevent the disease," says Dr. Flexner, "and the achievement of a cure, I may conservatively say, is not now far distant ... We have learned where it [the germ] resides, how the disease is spread, how the germ enters

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> the body, the main sources of infection and the means of combatting the disease."

Sir Macfarlane Burnet (Nobel Laureate 1960) wrote in 1949 (3):

> The natural way to prevent paralytic poliomyelitis is to ensure that infants are repeatedly infected with strains of virus of low paralytic potentialities. ... In a present day community, any use of living virus for immunization would be unthinkable. ... A killed virus is a theoretical possibility, ... but there is in sight no solution of the practical difficulties of producing it. Until someone can find a means of producing relatively large amounts of pure or nearly pure virus free of foreign nervous tissue, even experimental immunization of children would be absolutely unjustifiable. I can see no hope at present of such a vaccine being produced. ... I have adopted a frankly defeatist attitude toward the problem of poliomyelitis and I hope that future developments will prove me wrong. ... No means of controlling poliomyelitis is at present visible.

If only these two predictions had been switched in time—if Burnet's 1949 words had been spoken by Flexner in 1911 and vice versa— both predictions would have been correct, for it was in 1949 that Enders, Weller, and Robbins learned to grow large quantities of poliovirus in non-neural tissue. Successful polio vaccine came a few years later.

On Cardiovascular Diagnosis

In 1905 (the year that Korotkoff published his new non-invasive method for measuring both systolic *and* diastolic blood pressure, a method that at last permitted objective study of the natural course of hypertension and of methods to treat it) the *British Medical Journal*, official organ of the British Medical Association, argued that by sphygmomanometry "we pauperize our senses and weaken clinical acuity" (4).

James Mackenzie, England's great cardiologist, was antagonistic toward both the use of X-rays and the electrocardiogram because he was convinced that such aids would "blunt the senses and the acute perception of the clinical observer" (5).

The senior cardiologists of Great Britain strongly opposed and censured McMichael and Sharpey-Schafer in their pioneer efforts to use cardiac catheterization as a diagnostic tool (6). Nevertheless, by April 1945, the two had performed 353 cardiac catheterizations in humans without complications, apart from local venous thrombosis, and proved that all dire predictions of disaster were unwarranted.

On Cardiac Surgery

Sir Stephen Paget, author of the authoritative text, *The Surgery of the Chest*, predicted in 1896 (7):

> Surgery of the heart has probably reached the limits set by Nature to all surgery: no new method and no new discovery can overcome the natural difficulties that attend a wound of the heart. It is true that heart suture has been vaguely proposed as a possible procedure, and that it has been done on animals, but I cannot find that it has ever been attempted in practice.

On September 9 of that same year, Ludwig Rehn of Frankfurt performed the first successful suture of a human heart wound! A little later, Samways (1898), Brunton (1902), and Carrel (1912) all proposed methods to open the narrowed valve in patients with mitral stenosis. Sir Lauder Brunton (8) suggested that, before attempting this in man, surgeons should perform experiments on animals to develop a suitable technique. He himself obtained the necessary license and certificates for animal investigation and did a few preliminary experiments. However, his limited time prevented him from carrying on, and he decided to try to stimulate interest of fellow surgeons in continuing the work. His brief communication to the *Lancet* was followed the next week by an editorial in the same journal castigating him for suggesting such a highly dangerous procedure that might actually make a patient worse, even if he survived the operation.

Little more was done about the mitral valve until Cutler and Levine (1923) and Souttar (1925); each report dealt with one patient. After another long hiatus, Bailey performed a successful mitral commissurotomy in 1949, and surgery on cardiac valves began its spectacular journey.

Munro, in 1907, proposed ligating a patent ductus arteriosus, but as late as 1939, Robert Gross's professor cautioned him against performing such an operation. Despite this advice, Gross was bold enough to ligate such an artery and overnight became the father of modern cardiac surgery. Yet shortly thereafter, when a pediatric cardiologist named Helen Taussig proposed to Gross that he perform an aortic-pulmonary artery shunt to relieve cyanosis in "blue babies" with a type of congenital heart disease known as tetralogy of Fallot, he turned her down. Gross's crystal ball was working only 50% of the time in 1940, but he was then America's leading cardiac surgeon and would certainly have been chairman of a 1940 NIH Committee to advise on cardiac surgery.

According to Medawar (9), Lord Moynihan, Britain's great surgeon, said in 1930, "Surgery as a craft has reached its peak" and, in 1932, "We can surely never hope to see the craft of surgery made much more perfect than it is today. We are at the end of a chapter." Medawar noted in 1975 that Moynihan remarked that operations could be carried out (in 1932) of a kind that even Lister would not have dreamed possible, but the thought never entered Moynihan's head that in the future, operations could be done that even he considered impossible in 1932. Medawar commented that Moynihan was a very great surgeon and a man of very great gifts, but these did not include clairvoyance!

John Gibbon, who spent 19 years bringing the pump-oxygenator (artificial heart lung) from a "mission impossible" idea to a practical device now essential for much of modern cardiac surgery, was fortunate enough to get laboratory space in which to work and funds for technical help, first at the Massachusetts General Hospital and then at the University of Pennsylvania. But Gibbon recalls that "Churchill [in Boston] was not enthusiastic about my project but raised no objection to my pursuit of my goal. Still less enthusiastic was my friend, Walter Bauer. ... Eugene Landis was my only medical friend who did not discourage me ... he said that if I thought I could succeed, it was worth giving it a try" (10). Landis recalls encouraging Gibbon, all the while thinking (silently, thank heavens), "what a long, hard, impossible road to choose" (11).

When Gibbon worked in Ravdin's Department of Surgical Research at Penn (1936 to 1942), I was an instructor in Pharmacology working on the floor below Jack and Mary Gibbon. I know that they were well liked and also respected for their intense dedication to their project, but I don't recall any faculty member, old or young, who had much interest in it or expectation that anything would come of it. I can't think of any Presidential Advisory Panel, appointed in 1936 to 1942, that would have recommended "full speed ahead" on Gibbon's work.

On the Discovery of Insulin

Many scientists in the 30 years preceding 1921 tried to learn the function of the islets of Langerhans in the pancreas. So it is not remarkable that when a young small town surgeon, Frederick Banting, who had done *no* research (let alone research in the baffling field of endocrinology-metabolism) had an idea and took it to J. J. R. Macleod (Professor of Physiology at the University of Toronto, Canada's scientific leader in this field), Macleod did not roll out the red carpet. It appears that Macleod in fact did his best to discourage Banting. What did Banting know about the anatomy, histology, and physiology of the pancreas, about the chemistry of blood sugar, about clinical diabetes, and what could Banting hope to accomplish that highly trained physiologists had not succeeded in achieving, establishing, or proving? Banting replied that all he wanted was 10 dogs and an assistant for eight weeks. Macleod was about to go on vacation in Europe and against his better judgment provided Banting with 10 dogs and an assistant, Charles Best, who was a medical student. Banting started to work on May 16, 1921, and on July 27 (10 weeks later) administered the first crude preparation of insulin into a dying depancreatized dog. By early 1922, the purification and commercial preparation of insulin, and the saving of lives of dying diabetic patients, had become the full-time occupation of Banting, Best, Macleod, Collip (a chemist), and scores of others. In 1923 Banting and Macleod won the Nobel Prize.

Harry Eagle has stated that he has "more confidence in the collective judgment of 10,000 independent investigators than in that of 10 wise men sitting around a table" (12). In this case, Banting and Best were two of 10,000 scientists with special imagination, conviction, and determination, and Macleod was one of 10 wise men.

On Heparin

No one predicted an important use for heparin (discovered by McLean in 1916) until the 1940s. William Howell, McLean's professor, presented a Harvey Lecture in 1916-17; his only prediction with respect to the clinical importance of heparin was that hemophilia might be due to excessive amounts of heparin in circulating blood (13). It is surprising that he did not suggest that heparin be purified and used to replace hirudin in the artificial kidney (vividiffusion) reported by Abel, Rowntree, and Turner in 1913 and 1914; the best explanation is that no one in 1916 predicted any clinical use of the artificial kidney!

Best, co-discoverer of insulin in 1921, put Charles and Scott to work in 1933 to prepare pure heparin for laboratory use. Best also gave a Harvey Lecture on heparin; this one was in 1940 (14) after the Toronto group had pioneered in the clinical use of heparin. Best did "hope that some day the technique [of exchange transfusions] may be perfected by which kidneys or perhaps some other organ of normal subjects may be made available, as it were, to patients in whom there is a serious *but presumably transient* deficiency in some physiological function." He also said that "heparin is a valuable adjuvant in certain types of vascular surgery ... on the substitution of veins for arteries in certain cases of arterial aneurysm, the removal of emboli, ... and the repair of severed arteries." He also suggested carefully controlled clinical studies of the value of heparin in treating patients with coronary artery thrombosis.

Best, in 1940, was both cautious and accurate in most of his predictions, and from his association with Murray's surgical group in Toronto made some that didn't occur to Howell in 1916. But Best still did not predict one major use of heparin—as an anticoagulant for pump-oxygenators— although Gibbon had been at work on his artificial heart-lung for six years and had published papers in 1937 and 1939 mentioning his use of heparin.

On the Artificial, Implanted Heart

In mid-1964, one of America's most eminent scientists wrote to the Director of the National Heart Institute:

> For sometime I have been puzzling why an artificial heart has not yet been made available as a fruit of our technological developments. ... I believe the problem is technically difficult but easily manageable within the framework of our present scientific knowledge and technical proficiency. We have however permitted it

> to remain a subject of fragmentary scientific studies rather than of a unified engineering program. We do ourselves a great disservice to neglect the opportunity of a systems response to what is now a well-defined technical problem, which is so much a matter of engineering design, material development and empiric testing and should not be confused with the basic research that was needed at its foundation.

The NHI had indeed been supporting research and development on the artificial heart, but following this "we know all we need to know; it's time to apply it" letter from a Nobel Laureate, the National Heart Institute drew up a master plan for a systems approach to the artificial heart that called for the signing of contracts by July 1, 1965 and implantation of artificial hearts in patients by February 14, 1970. It asked for Congressional appropriations of $270,000 for fiscal year 1965, about $8,000,000 for 1966, and a little more than $100,000,000 in 1967—in true "moon shot" style.

By mid-1976, a clinical practical device was still not on the horizon and the NHI (now the NHLBI) had long since decided to emphasize support of short-term ventricular-assist devices rather than the more dramatic implanted plutonium-powered mechanical heart. Why? Some may say, "because Congress never appropriated the 'moon shot' type of money for an all-out systems approach for the plastic heart." Could it have been because we really did *not* know all we needed to know and that we needed more than a group of engineers to glue the parts together?

On the Management of Infection

Oliver Wendell Holmes, Professor of Anatomy and Physiology at Harvard Medical School, presented evidence in 1843 that childbed fever was contagious and was frequently carried from patient to patient by physicians and nurses. He was attacked by C. L. Meigs, Professor of Midwifery at Jefferson Medical College in Philadelphia and by Hodge, Professor of Obstetrics at the University of Pennsylvania:

> Hodge begged his students to divest their minds of the dread that they could ever carry the dreadful virus. Meigs scorned Holmes's deductions as characteristic of the "jejune and fizzenless vaporings of sophomore writers" and added that he preferred to attribute the deaths to "accident, or providence, of which I can form a conception, rather than to a contagion of which I cannot form any clear idea, at least as to this particular malady." (See 15, 16)

Undoubtedly, Holmes had antagonized members of the medical profession by suggesting that they were carriers of death, and his scientific observations further drew scorn because he was "a sophomore writer."

In 1847, Ignaz Semmelweiss of Vienna independently proposed that childbirth fever was contagious. He stated that it was carried to women in the city's obstetrical wards by "cadaveric material" on the hands of physicians and students coming directly from the postmortem room to the delivery room. He instituted a strict routine that all hands about to touch patients must first be thoroughly washed in a solution of chlorinated lime. Mortality from puerperal sepsis fell at once from 12 to 3 per cent and later to one per cent. But his revolutionary doctrine, like that of Holmes incriminated obstetricians as the carriers of disease and death and aroused fierce opposition from medical authorities, including the great pathologist, Virchow (16). Semmelweiss, ostracized, denounced and ridiculed, died 18 years later in an insane asylum. If there had been a Viennese Imperial task force on puerperal sepsis, would it have given Semmelweiss a chance to prove his theory?

Lord Lister's acceptance of Pasteur's germ theory of disease and his vigorous attempts to achieve antisepsis in surgical operating rooms met with fierce opposition partly by hospital administrators who saw the reputations of their hospitals being destroyed, and partly by physicians who still disbelieved the germ theory of infection. It is interesting that one physician most influential in disparaging Lister was James Simpson, who, when he introduced chloroform into obstetrics and surgery only a few years earlier, was denounced by the clergy, both from the pulpit and in pamphlets, on the grounds that anesthetics were decoys of Satan (see 17). Apparently, being attacked without reason does not deter one from attacking another without reason!

In 1938, the Medical Research Council of England turned down Ernst Chain's request for very modest support of his research project; on the chemical and biochemical properties of antibacterial substances produced by microorganisms (now known as *antibiotics*), which a few years later (with support from the Rockefeller Foundation) led to the purification of penicillin and a Nobel Prize; Medawar (9) recalls Lord Florey's telling him that wise old gray heads in England shook (or wobbled) regretfully from side to side, pronouncing that the future of antibacterial therapy lay not with extracts of fungi and other medieval-sounding concoctions but with synthetic organic chemicals of which sulfanilamide was the paradigm. We must flunk the MRC of England in clairvoyance on this decision; fortunate indeed that not *all* funds are controlled by one agency with one set of advisers!

Some Predictions in Other Sciences

Any scientist—not only biomedical scientists— can have a clouded crystal ball. Neils Bohr, Danish atomic physicist and Nobel Laureate, is reported to have said, "It is very difficult to predict —especially the future"; Mark Twain remarked that "the man with a new idea is a crank until the idea succeeds"; and Peter Medawar entitled one of his articles "Some Follies of Prediction."

Francis Bacon could not believe that the earth goes around the sun; Galileo could not accept Kepler's evidence that planets move in ellipses; Maxwell in 1879 said, "Atoms ... continue to this day as they were created, perfect in number, measure and weight"; Pasteur at the height of his fame stated that scientific methods could never be applied successfully to the study of emotions (at a time when Pavlov and Cannon were waiting in the wings); Lord Kelvin, throughout a very distinguished life in science, never discarded the concept that the atom is an indivisible unit (18, 19).

In 1845, J. J. Waterston submitted a paper on the molecular theory of gases to the Proceedings of the Royal Society. Although Waterston's work anticipated much of the later work of Joule, Clausius, and Maxwell, the referee entrusted with evaluating the manuscript said, "This paper is nothing but nonsense." The paper lay in oblivion until rediscovered 45 years later. According to Trotter (20), after the rejection Waterston lived on disappointed and obscure for many years and then disappeared, leaving no sign.

Thomas Edison, one of the world's greatest and most prolific inventors (he had almost 1,300 patents in all), could not predict any practical use for his only discovery in "pure" science. It was the ability of electrons, under certain conditions, to travel unimpeded through a vacuum. This "Edison effect" later was used by J. A. Fleming to develop a "current rectifier" and that in turn permitted Lee deForest to invent the first radio tube (see 21).

In 1882, Western Union commissioned a committee of experts to look into a new invention claimed to transmit the human voice from one place to another through wires. The inventor's name was A. G. Bell; he called his device the telephone. Western Union's blue ribbon panel reported, in part (22):

> This fellow Bell's profession is that of a voice teacher, particularly a teacher of the deaf. He appears to have had no previous experience with any form of communication electronic or otherwise; yet he claims to have discovered a concept which has been overlooked by hundreds of experts who have spent years in the particular field.
>
> Bell's proposal to place his instrument in every home and business is, of course, fantastic in view of the capital costs involved in installing endless numbers of wires.
>
> We feel that it is unlikely that any substantial number of people will ever buy such a concept in view of the fact that there are telegraph offices now giving efficient round-the-clock service in every neighborhood in even the smallest towns.
>
> In conclusion the committee feels that it must advise against any investment by Western Union in Bell's scheme. We do not doubt that it might find a few users in special circumstances—such as between the bridge of a ship and the engine room—but any development of the kind and scale which Bell so fondly imagines is utterly out of the question.

The opinion of Western Union's Commission was shared by P. G. Tait, Scottish physicist eminent in the fields of thermodynamics and gas kinetics, who, learning of the invention of the telephone, said, "It is all humbug, for such a discovery is impossible" (19).

Professor Simon Newcomb, famous American astronomer, professor of mathematics and astronomy at Johns Hopkins University and editor of the *American Journal of Mathematics*, said (23):

> "Human flight is not only impossible but illogical." A new metal or a new force in nature would have to be discovered before man could fly. Even if by some miracle a man should invent a power-machine that could get off the ground, it would inevitably crash and kill its pilot. "Once he slackened his speed, down he begins to fall. Once he stops, he falls a dead mass."

His views were published in 1903. On December 17 of that year, Orville and Wilbur Wright made their first flight in a heavier-than-air plane, at Kitty Hawk, North Carolina.

Stern (24) points out that:

> Most steel men knew that Henry Bessemer's ideas of making steel by blowing air through molten iron was absurd. Despite universal ridicule Bessemer built his own plant, and revolutionized steel making. The ridiculers are lost to history. William Siemens knew that his open-hearth process for making steel was fundamentally sound. No one else believed it until he had demonstrated its success in the steel plant he built himself. After the steel makers had all climbed on the wagon with him, many of them contended that they had thought of it first. Hadfield discovered that steels containing 13 per cent of manganese were strong, tough, and very desirable. He couldn't convince even his own father, a foundry owner, that this was true. Eventually, Hadfield's work led to the opening of the entire field of alloy steels. Not many years ago all electronic experts knew that chromium plating could never be practical—all but one or two. Millions of automobile bumpers show evidence of the experts' errors. Before 1908 it was known that it was impossible to make ductile tungsten wire, but in that year Dr. W. D. Coolidge, of the General Electric Laboratory, forced that tradition out of the window.

Success in Predicting

Some decisions that involved large sums of money and changed the directions of many scientists have been very wise ones. The decision of the National Foundation for Infantile Paralysis to put much of its resources into basic studies in vi-

rology rather than into specific applied programs is credited with speeding the work of Enders and his associates and the development of a successful and safe polio vaccine. The scientific advice that led the United States into the Manhattan project and the atomic bomb was correct and greatly increased the respect of Congress and the Administration for scientific consultants. As Senator Tydings admitted to Dr. Leo Szilard in 1945 (25):

> Doctor, if in 1939 we had been conducting a hearing like we are conducting today, and men like yourself had come before our committee and projected the possible development of the bomb up to now with reasonable accuracy, I imagine they would have been called a lot of crackpots and ... visionaries who were playing with theories. I certainly would not have had the receptivity that I have today to say the least.

The decision in the early 1940s of the Committee on Medical Research of the Office of Scientific Research and Development to proceed with a crash program for mass production of penicillin had far-reaching effects, and part of its success must be attributed to Roosevelt's decision to activate these advisory committees long before America entered World War II.

Many cooperative clinical trials, such as those for penicillin and antituberculosis drugs, were also highly successful. The decision of the Armed Forces Epidemiological Board to create a Streptococcus Disease Laboratory in 1948 resulted in the prophylactic treatment of acute rheumatic fever. It appears that special Task Forces, Panels, or Commissions can work very well when all the basic information is at hand, when the goal is clear cut, and when the time is ripe to wrap up a problem. They are less effective when the basic information is not at hand and one needs whole new ideas and concepts and methods for scientific advance.

No one has yet made a long-term, serious attempt to study the factors that make for successful predictions (26), although a recent Ciba conference, "The Future as an Academic Discipline," suggests the possibility that departments dealing with the Future may stand side by side with departments of History in the structure of universities (27).

Concluding Remarks

(1) This look into the Retrospectroscope makes no pretense at being a scientific, quantitative analysis of clairvoyance. But it does produce examples of prominent scientists who were sometimes deficient in this attribute—enough examples to justify a study of the fallibility or infallibility of scientists eminent enough to become advisers to Congress and institutes. Such a study is needed because leading scientists today control not only the direction that their own research takes, and maybe that of their immediate staff, but also the direction of research of many, perhaps the great majority, of biomedical scientists in the United States. How? Simply because Presidents, Congress, Institutes, and Foundations, eager to cure dread diseases as quickly as possible, have appointed Councils, Panels, Task Forces, Commissions, and Committees and asked the members of these to look into their crystal ball and find specific programs guaranteed to eliminate these diseases promptly. Quite often such panels recommend significant changes in federal research budgets that in effect decrease the free choice of scientists to do what they and their peer review groups consider to be good science.

(2) Producing examples of errors in predicting the future requires naming names. Naming scientists was certainly not the prime purpose of this Retrospectroscope, nor was it intended to lessen the stature of the named scientists. Obviously, this look backward would have been pointless if it dealt only with unheard of, unsuccessful scientists. As my kindly old professor of surgery said repeatedly in his quiz sections with students, "Boys, everyone makes mistakes; that's why they always put erasers at the end of lead pencils." Science progresses by continuously revising, extending, or completely overturning existing "facts" to create new concepts that may become "facts" and in turn be revised or discarded; it rarely progresses by producing "breakthroughs" on demand.

(3) This look has concerned itself with official clairvoyance at the national (Congressional or NIH) level that can alter the flow of vast amounts of research funds and so direct many scientists into fewer and more restricted channels. It has not concerned itself with *speculation* that, I believe, should be an important part of every oral or written scientific presentation. Such speculation, in my view, *broadens* the choice of individual scientists who are exposed to it because they are quite free to ignore it, verify it, or demolish it.

Julius H. Comroe, Jr.

REFERENCES

1. Comroe, J. H., Jr.: Retrospectroscope. How to succeed in failing without really trying. Am Rev Respir Dis, 1976, 114, 629-634.
2. New York Times, March 9, 1911, quoted by Paul, J. R.: A History of Poliomyelitis, Yale University Press, New Haven, 1971, p. 116.
3. Burnet, F. M.: Some aspects of the epidemiology of poliomyelitis. Proc R Australasian Coll Physicians, 1949, 4, 95-100.
4. British Medical Journal, 1905, quoted by Waterline, G. E.: From Bright toward light: The story of hypertension research. Circulation, 1962, 26, 1-6.
5. McMichael, J.: A Transition in Cardiology: The Mackenzie-Lewis Era. The Harveian Oration of 1975. Royal College of Physicians, London, 1976.
6. Cournand, A.: Cardiac catheterization. Acta Med Scand [Suppl], 1975, 579, 3-32.
7. Paget, S.: The Surgery of the Chest, J. Wright & Co., Bristol, 1896, p. 121.
8. Brunton, L.: Preliminary note on the possibility of treating mitral stenosis by surgical methods. Lancet, 1902, 1, 352.

9. Medawar, P. B.: Some follies of prediction, Hosp Practice, 1975, 10 (4), 73-74.
10. Gibbon, J. H., Jr.: The development of the heart-lung apparatus. Rev Surg, 1970, 27, 231-244.
11. Landis, E. M.: Personal communication, October 17, 1970.
12. Eagle, H.: Minutes of the Committee on Science Policy for Medicine and Health, Institute of Medicine, National Academy of Sciences, Washington, D. C., June 22, 1973.
13. Howell, W. H.: The coagulation of the blood. Harvey Lect, 1916-17, 12, 272-323.
14. Best, C. H.: Heparin and thrombosis, Harvey Lect, 1940-41, 36, 66-90.
15. Cannon, W. B.: The Way of An Investigator. A Scientist's Experiences in Medical Research, Hafner, New York, 1968 (facsimile of 1945 edition).
16. Stern, B. J.: Historical Sociology. Selected Papers of Bernhard J. Stern, Citadel Press, New York, 1959, pp. 363-365.
17. Ibid., p. 368.
18. Barber, B.: Resistance of scientists to scientific discovery, Science, 1961, 134, 596-602.
19. Stevenson, L.: Scientists with half-closed minds, Harper's, November 1958, 217, 67-71.
20. Trotter, W.: The Collected Papers of Wilfred Trotter, Oxford University Press, Humphrey Milford, London, 1941, p. 186.
21. Asimov, I.: Introduction. Of what use, in *The Greatest Adventure: Basic Research That Shapes Our Lives*, E. H. Kone and H. J. Jordan, ed., Rockefeller University Press, New York, 1974, pp. 1-7.
22. O'Flaherty, T.: To everything a season, San Francisco Chronicle, March 6, 1972, p. 40.
23. Untermeyer, L.: Makers of the Modern World. Wilbur Wright and Orville Wright. Simon and Schuster, New York, 1955, pp. 360-367.
24. Stern, p. 97 (see reference 16).
25. Atomic Energy: Hearings before the Special Committee on Atomic Energy, U. S. Senate, 79th Congress, 1st Session, U. S. Government Printing Office, Washington, D. C., 1945, p. 290.
26. Lilley, S.: Can prediction become a science?, Discovery, November 1946, pp. 336-340, reprinted in *The Sociology of Science*, B. Barber and W. Hirsch, ed., Free Press of Glencoe, New York, 1962, pp. 142-152.
27. The Future as an Academic Discipline, Ciba Foundation Symposium No. 36 (New Series), Elsevier, Amsterdam, 1975.

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Best Review Paper

Best Review Paper Published from November 1994 to November 1995 ($500)

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The Impact of a Postoperative Oxygen Therapy Protocol on Use of Pulse Oximetry and Oxygen Therapy—JJ Komara Jr, and JK Stoller [Respir Care 1995;40(11):1125-1129] ($500)

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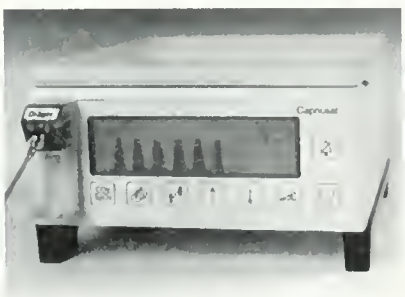

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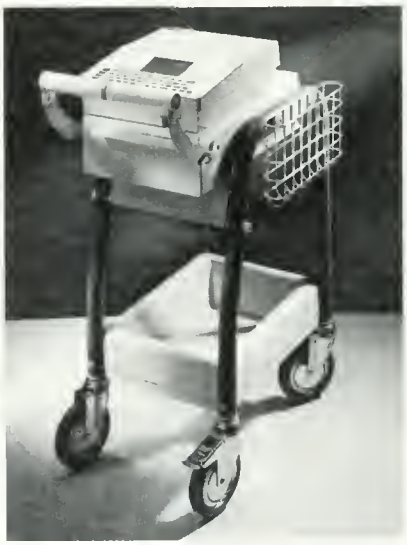


ELECTROCARDIOGRAPH RECORDER. Burdick Inc's new line of electrocardiograph (ECG) recorders are designed to use less space and provide higher efficiency. Burdick claims that its new Eclipse ECG recorders improve efficiency with more features such as a print preview function, high-quality tracings, ready-to-file reports, and easy data entry. The Eclipse is available in 6 configurations. Portable units with rechargeable batteries are also available. In addition, a new lightweight ECG cart designed for the Eclipse is offered. For more information, call Burdick Inc at (800) 955-5177, ext 205. Remember to mention RESPIRATORY CARE.

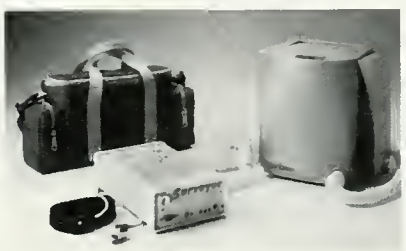
FINGER PULSE OXIMETER. Nonin Medical Inc introduces the Nonin ONYX™—a self-contained finger pulse oximeter. The new design, according to the manufacturer, incorporates batteries, electronics, and LED display into a unique finger sensor. The ONYX weighs 2 ounces including the batteries. The oximeter may be used for spot checks or attended monitoring in adult or pediatric patients. Write to Nonin Medical Inc, Dept RC, 2605 Fernbrook Lane North, Plymouth MN 55447-4755, for more information. Please mention RESPIRATORY CARE when you call (800) 447-ONYX (6699).

ASTHMA VIDEO. HealthScan Products, a division of Healthdyne Technologies, launches a new patient education video, *Living with Asthma: What You Can Do*. The video is divided into two, 6-minute segments, according to the producer. The first segment teaches patients how

to use the company's Personal Best® Peak Flow Meter; the second segment instructs patients in the use of the OptiHaler® Drug Delivery System. The video explains peak expiratory flow and measuring by the 3-zone method. In addition, patients are taught techniques to use with a metered-dose inhaler and spacer. Available in Spanish and English versions, the video is free to physicians and respiratory care professionals who use HealthScan Products. For more information, call (800) 962-1266. Please mention RESPIRATORY CARE when calling.

ASTHMA MONITOR. ENACT Health Management Systems announces the new pocket-size AirWatch™, available to patients via prescription. According to the manufacturer, the monitor uses low-cost microprocessor technology to record and store a patient's lung function data—peak expiratory flow and FEV_1. A friendly icon named Coach Wilby indicates an all clear, caution, or red zone based upon a percentage of the patient's personal best. Once the measurements are completed the patient may connect the monitor to a telephone jack and transmit the data to AirWatch Central™, ENACT's database center. This information is then faxed directly to the patient's physician in report form; the system also produces a monthly report. Physicians may use the information for immediate intervention, to evaluate the effectiveness of drug therapies, or to determine whether patients are following prescribed regimens. For more information about the monitor, call (800) 267-9452. Please mention RESPIRATORY CARE when calling.

TEMPERATURE RECORDER. Olympic Medical provides the Model 90 Olympic Temperature Recorder that can also print a record of every pasteurizing cycle for all Olympic Pasteurmatics. According to Olympic, the recorder continuously monitors water temperature and prints out the date, time, and water temperature (° C or ° F) during the pasteurization cycle. Other features include a large digital display with easy-to-read numbers, a support post for easy mounting on either side of the pasteurizer, and easy access to printout paper—standard adding machine tape. Please mention RESPIRATORY CARE when calling (800) 426-0353 for details. Write to Olympic Medical, Dept RC, 5900 First Ave So, Seattle WA 98108.



MONITORING SYSTEM. DeVilbiss offers the Surveyor™, a new monitoring system that works with nasal CPAP to provide a one-night respiratory-event study for patient follow-up. According to the company, the Surveyor works in conjunction with the DeVilbiss Horizon™ nasal CPAP system and records the time the patient is at pressure and breathing, for objective information about at-home CPAP adherence. The Surveyor records an 8-hour respiratory-event study, separate channels for flow, apnea/hypopnea, snoring, pressure, S_{pO_2}, and body position. The data can be downloaded to a computer using Windows software. In addition, the Surveyor can be used in the patient's home with a body position sensor and belt and an oximetry finger probe. Contact DeVilbiss at (800) DeV-1988 for details. Don't forget to mention RESPIRATORY CARE.

LABORATORY PROFICIENCY TEST. Accutest offers a proficiency test designed for respiratory and cardiopulmonary laboratories. The program, according to Accutest, helps labs meet regulatory requirements mandated by the Clinical Laboratory Improvement Amendment (CLIA) of 1988 and provides meaningful comparative statistics that allow labs to assess blood-gas, CO-Ox, and electrolyte-test accuracy. The service includes written notification of upcoming test deadlines, 21-day turnaround of performance reports, and supplemental test kits that help verify and document the effectiveness of corrective actions. For details, write to Accutest PO Box 999, Dept RC, Westford MA 01886, or call (800) 356-6788—please mention RESPIRATORY CARE.

RESPIRATORY CARE

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Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

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Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

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Article in a journal carrying pagination throughout volume:

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Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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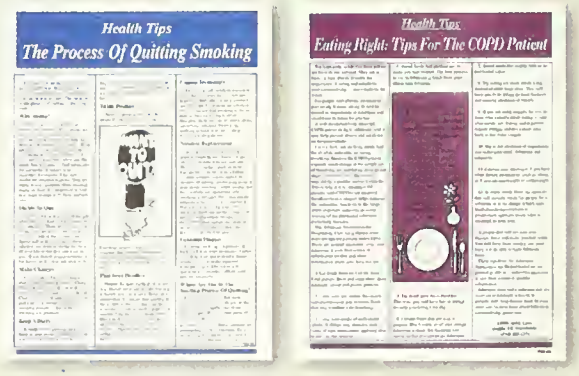

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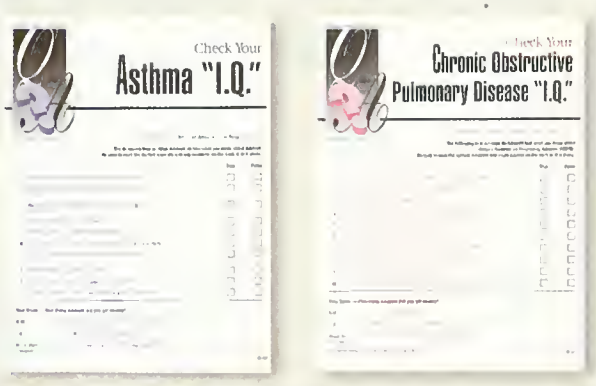

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The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC Annual Meeting in San Diego, California, November 3-6 1996. Accepted abstracts will be published in the October 1996 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation.

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3. Sex
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or
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☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other:

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other:

5. Expiration date (mo/day/yr)

6.
model #
catalog #
serial #
lot #
other #

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name, address & phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 (6/93)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 for more information or to report quality problems
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send your comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to

Reports Clearance Officer PHS
Hubert H. Humphrey Building
Room 721-B
200 Independence Avenue, S.W.
Washington, DC 20201
ATTN: PRA

and to
Office of Management and Budget
Paperwork Reduction Project
(0910-0230)
Washington, DC 20503

Please do NOT return this form to either of these addresses.

FDA Form 3500-back

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Food and Drug Administration
Rockville, MD 20857

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Calendar of Events

AARC & AFFILIATES

January 31–February 2 in Clackamas, Oregon. The OSRC announces its annual Pacific Northwest Respiratory Care Conference at the Monarch Hotel. Presentations include "Health Care Reform: What It Means to the Practitioner," "New Modes of Mechanical Ventilation," "What's Snooze in Sleep," and "Humor and Healing in Respiratory Care." CRCE credit has been requested. Contact Irene Iwata-Morgan at (503) 494-6158.

February 20–23 in Reno, Nevada. The American Lung Association of Nevada and the NSRC host the 15th Annual High Sierra Critical Care Conference at the Peppermill Hotel and Casino. The conference covers critical care topics in adult, pediatric, and neonatal medicine. Contact Sherry Landis, ALA of Nevada, PO Box 7056, Reno NV 89510.

March 13–15 in Southshore Lake Tahoe, California. Chapter 9 of the CSRC presents the 18th Annual Tahoe Conference, "Setting the Sails into the Winds of Change," at the Embassy Suites. CRCE credit has been requested. Contact Kathy Gallego at (510) 947-5375 or George Rice at (510) 947-5292.

May 1–June 30—Management Training Institute. The deadline to enroll in Module 2 (Communication Skills and Managerial Accounting Part 1) and Module 6 (Business and Health Law and Organizational Behavior) of the Management Training Institute (MTI) Independent Study Program is April 1. MTI is an 8-module program of the AARC and Indiana University. Sign up now to earn the MTI Certificate in Management Development. Call Barbara Hakes at (317) 274-4475.

OTHER MEETINGS

February 25–28 in Snowbird, Utah. The American College of Chest Physicians (ACCP) hosts the meeting on Cardiopulmonary Wellness and Rehabilitation. The meeting is approved for 12 hours of CRCE credit. Contact Renata Simonsen, ACCP Education Department, 3300 Dundee Rd, Northbrook IL 60062, (708) 498-1400, fax (708) 498-5460.

March 13–14 in Baltimore, Maryland. The Johns Hopkins Hospital Department of Respiratory Care presents a 2-day seminar, "Lectures in Adult and Pediatric Respiratory Care," at Johns Hopkins University School of Medicine's Turner Auditorium. Topics include sleep disorder breathing, pulmonary management of acute lung injury, nitric oxide, Joint Commission preparation, and liquid ventilation. The seminar is approved for 12 hours of CRCE credit. Contact Debora Brown at (410) 955-9276.

March 28–30, 1996 at Big Sky, Montana. The American Lung Association of Montana announces the 15th Annual Big Sky Pulmonary & Critical Care Medicine Conference. Contact the ALA of Montana, 825 Helena Ave, Helena MT 59601, (406) 442-6556, fax (406) 442-2346.

April 9–15, 1996 in Miami, Florida. The Ventilation Assisted Children's Center (VACC) of Miami Children's Hospital Division of Pulmonology announces its free camp for ventilation-assisted children and their families. Activities include field trips, swimming, games, arts, and crafts. The application deadline for overnight campers is January 5, 1996. Contact Director Moises Simpser or Coordinator Cathy Klein, VACC, Division of Pulmonology, Miami Children's Hospital, 3200 SW 60th Ct, Suite 203, Miami FL 33155-4076, (305) 662-VACC, fax (305) 663-8417.

September 18–21, 1996 in San José, Costa Rica. The Costa Rica Society for Respiratory Therapy presents the 2nd Latin America Congress and Caribbean Congress for Respiratory Therapy. Contact Lic. Carlos E Pereira Hidalgo, Apartado 184-1017, San José 2000, San José, Costa Rica, (506) 232-8310, fax (506) 232-7786.

MARK YOUR CALENDARS!

This year's AARC Convention

November 3-6, 1996 (Sunday–Wednesday)

San Diego, California

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B. ❑ Cardiopulmonary
C. ❑ Anesthesia Service
D. ❑ Emergency Dept

III. Specialty
1. ❑ Clinical Practice
2. ❑ Perinatal Pediatrics
3. ❑ Critical Care
4. ❑ Clinical Research
5. ❑ Pulmonary Function Lab
6. ❑ Home Care/Rehab
7. ❑ Education
8. ❑ Management

IV.Position
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B. ❑ Chief Therapist
C. ❑ Supervisor
D. ❑ Staff Technician
E. ❑ Staff Therapist
F. ❑ Educator
G. ❑ Medical Director
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E. ❑ Staff Therapist
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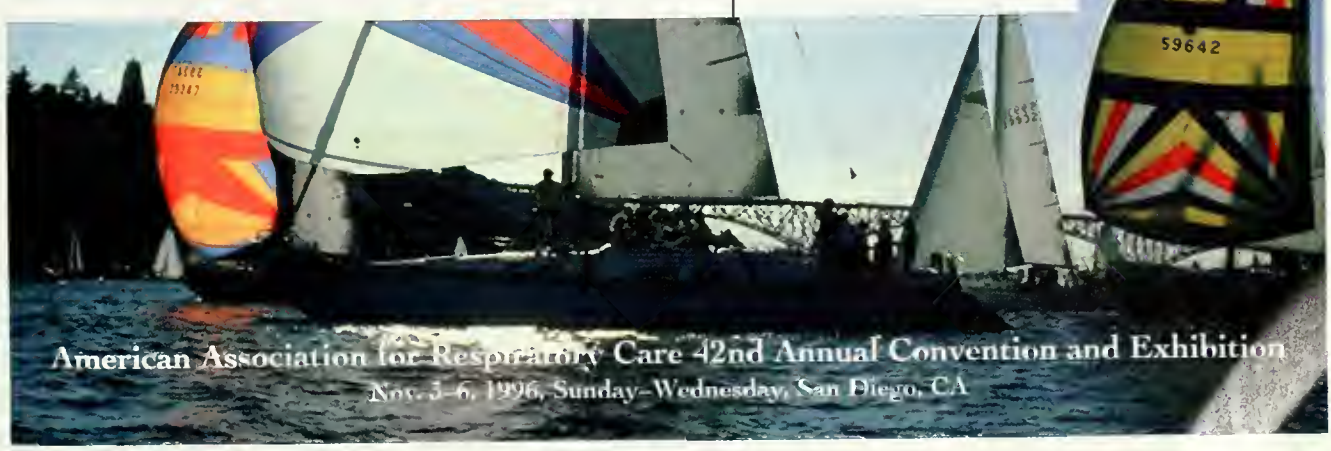
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July 12–14

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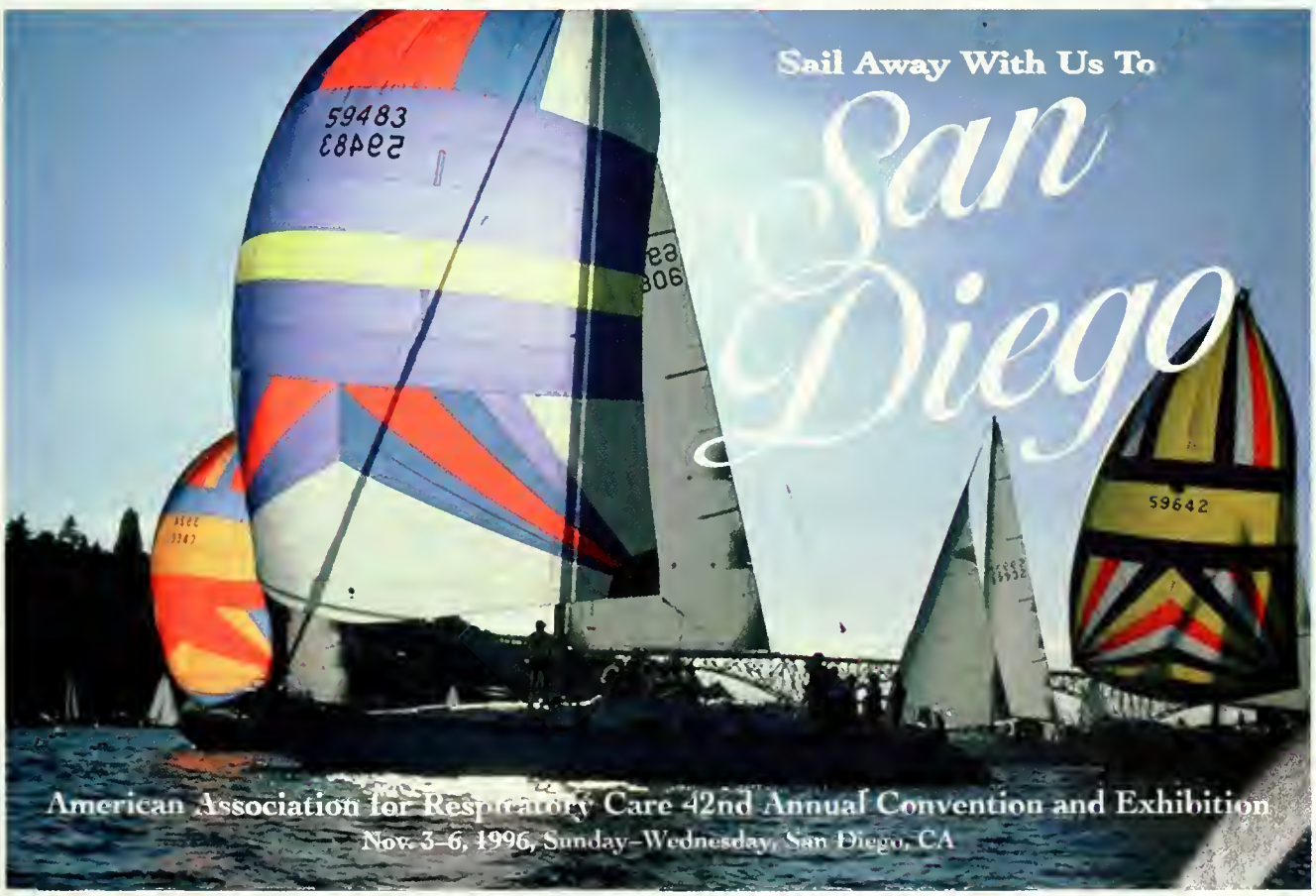
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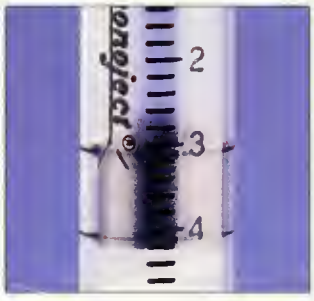

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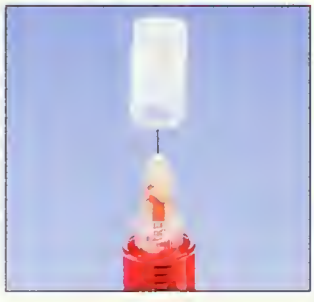

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